EFFECT OF NONSURGICAL PERIODONTAL THERAPY ON C-REACTIVE PROTEIN AND IRON INDICES IN HAEMODIALYSIS PATIENTS WITH CHRONIC PERIODONTITIS

DISSERTATION

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ENDORSEMENT BY THE PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"Effect of nonsurgical periodontal therapy on C-reactive protein and iron indices in haemodialysis patients with chronic periodontitis"** is a bonafide research work done by **Dr. Sheethel Menon V** under the guidance of **Dr. Arun Sadasivan, M.D.S**, Professor, Department of Periodontics, Sree Mookambika Institute of Dental Sciences, Kulasekharam.

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CONTENTS

LIST OF ABBREVIATIONS

AAP	-	American Academy of Periodontology
BOP	-	Bleeding on Probing
CAL	-	Clinical attachment level
CAPD	-	Continuous ambulatory peritoneal dialysis
CDC	-	Centre for Disease Control and Prevention
CKD	-	Chronic Kidney Disease
Cre-S	-	Serum creatinine levels
CRP	-	C - reactive protein
CVD	-	Cardiovascular disease
DMFT	-	Decayed, Missing or Filled Teeth index
DN	-	Diabetic nephropathy
ESA	-	Erythropoiesis-stimulating agents
ESR	-	Erythrocyte Sedimentation Rate
ESRD	-	End Stage Renal Disease
eGFR	-	Estimated Glomerular Filtration Rate
GI	-	Gingival index
GR	-	Gingival recession
Hb	-	Haemoglobin
HDL	-	High Density lipoprotein
IL-1β	-	Interleukin-1 beta
KDIGO	-	Kidney Disease: Improving Global Outcomes
K/DOQI	-	Kidney Disease Outcomes Quality Initiative
LDL	-	Low density lipoprotein
LPA	-	Loss of periodontal attachment

NHANES	-	National Health and Nutrition Examination Survey
NSPT	-	Non-surgical periodontal therapy
OHI-S	-	Oral hygiene index- Simplified
PAI-1	-	Plasminogen-Activator Inhibitor-1
PD	-	Peritoneal Dialysis
PI	-	Plaque index
PPD	-	Probing pocket depth
S-OC	-	Serum osteocalcin
TDI	-	Total dental index
TIBC	-	Total iron-binding capacity
TNF-α	-	Tumor Necrosis Factor-alpha
TSAT	-	Transferrin saturation
U-DPD	-	Urinary deoxypyridinoline

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ABSTRACT

Background

Periodontitis is a chronic inflammatory disease which leads to the destruction of the supporting tissues of the teeth. Interaction between periodontal pathogens and host immune response can trigger inflammatory mediators. These inflammatory cytokines have been reported to decrease the release of erythropoietin from kidney and thereby can lead to anemia. Periodontitis is found to increase the systemic inflammatory burden possibly facilitated by acute phase reactants like C-reactive protein (CRP), which aggravates the existing metabolic conditions like diabetes, hypertension and anemia in chronic kidney disease patients undergoing haemodialysis. Evaluation of anemia associated with chronic diseases can be done by the estimation of serum iron, total iron binding capacity, transferrin saturation and serum ferritin.

Aim of the study

To evaluate oral health status, clinical, renal and haematological parameters in systemically healthy chronic periodontitis patients and chronic kidney disease patients undergoing haemodialysis of varying duration and to evaluate effect of non-surgical periodontal therapy on clinical, renal and haematological parameters at baseline and 3 months postoperatively.

Materials and Methods

This was a comparative interventional study which included sixty chronic periodontitis patients. These chronic periodontitis patients were divided into three groups which consisted of chronic kidney disease patients undergoing haemodialysis for less than a year (Group I), chronic kidney disease patients undergoing haemodialysis for more than a year (Group II) and systemically healthy chronic

periodontitis patients (Group III). Clinical parameters were recorded at baseline, 1 month and 3 months after nonsurgical periodontal therapy. C reactive protein (CRP), transferrin saturation (TSAT) and serum ferritin was observed at baseline and 3 months after nonsurgical periodontal therapy.

Results

The clinical parameters like Plaque index, Gingival index, Bleeding on Probing, Probing Pocket Depth and Clinical Attachment level showed pronounced reduction at the end of 3 months in all the groups. CRP reduced in all the three groups 3 months post-operatively, but statistically significant reduction was seen in Group I and group III only. TSAT increased significantly in all the three groups. With regard to serum ferritin, it was seen that serum ferritin increased significantly in Group I and Group II, whereas significant reduction was seen in Group III.

Conclusion

It was seen that clinical parameters reduced in both systemically healthy periodontitis patients as well as chronic periodontitis patients undergoing haemodialysis of varying duration. It was noted that nonsurgical periodontal therapy can bring about reduction in CRP level and also improvement in iron indices in systemically healthy periodontitis patients and also in chronic kidney disease patients undergoing haemodialysis of varying duration. However studies with larger sample size and longer duration of follow up are required to confirm the findings.

INTRODUCTION

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with increased probing depth formation, recession, or both.[1] The microorganisms which are responsible for initiation and progression of periodontal diseases colonize the tooth surface and also at/or below the gingival margin and epithelial surfaces.[2] There is a wide variation in the bacterial species in the subgingival plaque. Plaque induced gingival disease generally consist of gram positive aerobic species, whereas there is a predominance of gram negative species in plaque samples of chronic periodontitis patients with almost 85% anaerobic or facultative anaerobic species.[3]

Biofilm formation and its persistence on the tooth surface present a challenge to the oral, sulcular and junctional epithelia.[4] The host epithelial cells are triggered by bacterial enzymes, endotoxins and exotoxins, bacterial waste products and surface components which secrete proinflammatory cytokines. Host immune response to these bacteria in biofilm is by the cooperation of innate and acquired immune system. Even though periodontitis is a chronic inflammatory disease, agents of acute phase of inflammation like C-reactive protein (CRP), plasminogen-activator inhibitor 1(PAI-1) and fibrinogen, which belong to innate immune system are also involved in the disease. This cause the activation of complement system, neutralization of pathogenesis agents, stimulation of repair system and degeneration of different tissues.[5] The relationship between periodontitis and systemic disease(periodontal medicine) is a two-way relationship with systemic host factors acting locally to affect periodontal destruction and the local bacterial challenge generating a widespread

effects with the potential to induce adverse systemic outcome.[6] Periodontal disease lead to a transient increase in the circulating levels of Interleukin -1β (IL-1β), Tumor Necrosis Factor- α (TNF-α) and Prostaglandin E_2. This can be considered as contribution of periodontal disease to systemic inflammation.[7]

Kidney performs four essential functions like excretion of end products of metabolism (like urea), regulation of blood volume and electrolyte concentration, regulation of erythrocyte production in the bone marrow through the secretion of erythropoietin and participation in calcium homeostasis through hydroxylation of vitamin D_3 into active or inactive metabolites.[8] Renal function can be assessed by the measurement of Glomerular Filtration Rate (GFR). Chronic Kidney Disease (CKD) is defined according to the presence or absence of kidney damage and the level of kidney function-irrespective of the type of type of kidney disease. The normal level of GFR may vary based on the age, gender and body size. The normal mean GFR for young adults is approximately 120-130mL/min/1.73m^2.[9] All the individuals with GFR <60mL/min/1.73m^2 for ≥3 months are classified as having chronic kidney disease, irrespective of the presence or absence of kidney damage. Chronic renal failure is characterized by a gradual reduction in the number of functional nephrons. Due to progressive renal damage, the excretion of body metabolic waste products is impaired, resulting in a state of intoxication called uremia, which is characterized by increased levels of acute phase proteins(like CRP), certain cytokines, and even macrophages.[10] Chronic renal failure can lead to terminal or End stage renal disease(ESRD). In these patients kidney function has deteriorated to a point where body suffers chronic systemic abnormalities. In such cases renal replacement therapy is required in the form of dialysis and/or kidney transplantation.[11]

Patients with ESRD are at higher risk of developing cardiovascular complications like atherosclerosis due to factors like diabetes, hypertension, dislipidemia, inflammation, malnutrition, and predisposition to infection.[12-14] Among the several associated factors of atherosclerosis, more attention has been given to the contribution of inflammation and its consequence in haemodialysis patients.[15] The causes of inflammation in haemodialysis patients are multifactorial. The patients on dialysis have an increased production of TNF-α, IL-6, IL-10 and IL-12 due to the presence of inflammatory processes.[16] Inflammatory reaction may originate from several sites including graft or fistula infections, bioincompatible dialysis membrane, dialysate, endotoxin exposure, back filtration, chronic infections, and malnutrition.[17] Poor oral, dental and periodontal health in haemodialysis patients can be a source of inflammatory reactions.[18, 19.]

Inflammation is closely related to protein–energy malnutrition in dialysis patients.[20] Serum ferritin concentration and transferrin saturation(TSAT) ratio are among the two most commonly used markers of iron status in maintenance dialysis patients.[21] The serum ferritin reflects storage iron, and absolute iron deficiency, according to the Kidney Disease Outcomes Quality Initiative(K/DOQI) guidelines, correlates with serum ferritin <100 ng/ml.[22] The TSAT is the serum iron divided by the total iron-binding capacity (TIBC), which corresponds to circulating iron. The TIBC reflects transferrin, the protein to which virtually all iron in the blood is bound. The K/DOQI workgroups have determined that absolute iron deficiency, the absence or near absence of stainable iron in the bone marrow, correlates with TSAT <20% and that there is a risk for iron overload when the TSAT exceeds 50%. It has been shown that patients on haemodialysis have worse oral condition

compared to general population, including increased prevalence for periodontitis.[23-26]

The cornerstone of periodontal therapy is nonsurgical management which include scaling and root planing.[27] One of the aims of Non-surgical periodontal therapy (NSPT) is to restore gingival health by completely removing elements that provoke gingival inflammation (which includes biofilm, calculus and endotoxins) from the tooth surface. NSPT have shown to reduce the number of subgingival microorganisms and also provide a shift in the composition of subgingival biofilm from one with higher number of gram negative anaerobes to one dominated by gram positive facultative bacteria compatible with health.[1] NSPT have shown to improve the systemic inflammatory markers in patients with periodontitis.[28-31]

Since there is higher prevalence of oral health disorders and also strong associations among chronic periodontitis, systemic inflammation and adverse outcomes in haemodialysis patients, more studies on periodontal therapeutic interventions are warranted. Therefore this study was designed to evaluate the effect of nonsurgical periodontal therapy (before and 3 months after) on clinical parameters[Bleeding on Probing (BOP), Plaque index (PI), Gingival index (GI), Probing pocket depth (PPD) and Clinical Attachment Level (CAL)], C-reactive protein and renal parameters (which include Serum ferritin, TSAT, serum albumin, serum creatinine, Haemoglobin, ESR, GFR) in haemodialysis patients with chronic periodontitis and compare the results with that of systemically healthy chronic periodontitis patients.

AIMS & OBJECTIVES

1. To evaluate oral health status, clinical parameters, vital signs, renal and haematological parameters in systemically healthy chronic periodontitis patients and chronic periodontitis patients with chronic kidney disease undergoing haemodialysis of varying duration.
2. To assess the effect of non-surgical periodontal therapy on clinical parameters at baseline, 1 month and 3 months postoperatively.
3. To assess the effect of nonsurgical periodontal therapy on renal and haematological parameters at baseline and 3 months postoperatively.

REVIEW OF LITERATURE

Kidney is composed of nearly one million anatomical and functional units which are called the nephrons.[39] When a nephron is destroyed, it is unable to regenerate and therefore kidneys compensate by hypertrophy until half of the existing nephrons have been destroyed. Once this occurs, renal functional impairment starts to appear. Chronic kidney disease is characterized by a gradual reduction in the number of the functional nephrons which lead to progressive decrease in kidney function to renal failure. Proteinuria and hematuria are the most commonly used markers of kidney damage.[40] There are several risk factors for chronic kidney disease which include age>60 years, diabetes, hypertension, obesity, macroalbuminuria, smoking, C-reactive protein, elevated total cholesterol.[41]

Chronic kidney disease is often complicated by multiple infections which is mostly due to impairments in both specific and nonspecific immune response.[26] Cardiovascular disease (CVD)- related events are the main reason for mortality in Chronic kidney disease patients.[42] Systemic inflammation is considered as a non-traditional risk factor for increased CVD events in such patients. When kidney function deteriorates, dialysis or renal transplantation is needed. Chronic kidney disease is defined as abnormalities of kidney structure or function, present for more than 3 months, with implications for health.[43] The Kidney Disease: Improving Global Outcomes (KDIGO) organization developed clinical practice guidelines in 2012 which classified chronic kidney disease based on cause, GFR category and albuminuria category. Identifying the cause is emphasized due to its fundamental importance in predicting the outcome and guiding choice of cause specific treatment.

GFR categories have been mentioned below.

G1: normal eGFR ≥ 90 mL/min per 1.73 m^2

G2: eGFR between 60 to 89 mL/min per 1.73 m^2

G3a: eGFR between 45-59 mL/min per 1.73 m^2

G3b: eGFR between 30-44 mL/min per 1.73 m^2

G 4: eGFR between 15 to 29 mL/min per 1.73 m^2

G5: eGFR< 15 mL/min per 1.73 m^2 or end-stage renal disease (ESRD).

Three albuminuria categories were proposed for simplification and initial assessment and prognostication.

Albuminuria	**Albumin excretion rate**	**Albumin-creatinine ratio**	**Descriptor**
A1	<30	<30	Normal to mildly increased
A2	30-300	30-300	moderately increased
A3	>300	>300	severely increased

End stage renal disease (ESRD) (GFR < 15 mL/min per 1.73 m^2 is fatal without renal replacement, which can be provided by dialysis (haemodialysis or peritoneal dialysis) or renal transplantation.

Periodontitis is a chronic inflammatory disease that leads to the destruction of periodontal tissue which is characterized by pocket formation, recession or both.[40] Severe periodontitis is the sixth most common human disease which causes micro-ulceration of the investing sulcular and pocket lining epithelium of the affected teeth.[44] The systemic inflammatory response seen in periodontal disease has several

components which includes dissemination of periodontal pathogenic bacteria, their products and locally produced inflammatory mediators. Acute phase reactants are also found to be increased in periodontal disease.[45] Therefore periodontal disease can in turn add to the chronic inflammation present in chronic kidney disease.

Prevalence of Chronic Kidney Disease

Kaze FF et al, 2015[46]conducted a study to find the prevalence and determinants of CKD and albuminuria in urban and rural adults Cameroonians. This was a cross-sectional study of 6-month duration, conducted in the health district of Dschang (Western Region of Cameroon), using a multistage cluster sampling. All adults diagnosed with albuminuria (≥30 mg/g) and/or decreased estimated GFR (<60 ml/min/1.73 m^2) were re-examined three months later. Logistic regression models were used to relate baseline characteristics with prevalent CKD. Study included 439 participants with a mean age of 47 ± 16.1 years; with 185 (42.1 %) being men and 119 (27.1 %) being urban dwellers. There was a high prevalence of hypertension (25.5 %), diabetes (9.8 %), smoking (9.3 %), alcohol consumption (59.7 %), longstanding use of herbal medicine (90.9 %) and street medications (87.5 %), and overweight/obesity (53.3 %) which were predominant in rural area. The prevalence of CKD was 13.2 % overall, 14.1 % in rural and 10.9 % in urban participants. Equivalents figures for CKD stages G3-G4 [G3a(eGFR 45–59); G3b (eGFR 30–44); G4 (eGFR 15–29)] and albuminuria were 2.5 %, 1.6 % and 5.0 %; and 12.1 %, 14.1 % and 6.7 % respectively. Existing hypertension and diabetes were associated with all outcomes. Elevated systolic blood pressure and the presence of hypertension and diabetes were the predictors of albuminuria and CKD while urban residence was associated with CKD stages G3-G4. They concluded that the prevalence of CKD

and albuminuria was high in this population, predominantly in rural area, and driven mostly by the commonest risk factors.

Anupama YJ et al, 2014[47] conducted a study to find the prevalence of CKD in urban population near Shimoga, Karnataka and to study the risk factor profile. Door-to-door screening of 2091 people aged 18 and above was carried out. Demographic and anthropometric data were obtained, urine was analyzed for protein by dipstick and serum creatinine was measured in all participants. eGFR was estimated using the modification of diet in renal disease (MDRD) equation and Cockcroft‑Gault equation corrected to the body surface area (CG-BSA). The total number of subjects studied was 2091. Mean age was 39.88 ± 15.87 years of which 45.57% were males. The prevalence of proteinuria was 2.8%. CKD was seen in 131 (6.3%) subjects when GFR was estimated by MDRD equation. The prevalence of CKD was 16.54% by the CG-BSA method. There was a statistically significant relationship of CKD with gender, advancing age, abdominal obesity, smoking, presence of diabetes and hypertension. They concluded that the prevalence of CKD is higher compared to that from rural India and is comparable to from the urban Indian populations. The wide difference between the CKD prevalence between MDRD and CG‑BSA equations suggests the need for a better measure of kidney function applicable to Indian population.

Singh KA et al, 2013[48] conducted a study to determine the prevalence and risk factors for CKD in India. This was a cross-sectional study which screened 6120 Indian subjects from 13 academic and private medical centres all over India. The total cohort included in this analysis was 5588 subjects. The mean ± SD age of all

participants was 45.22 ± 15.2 years (range 18–98 years) and 55.1% of them were males and 44.9% were females. They also obtained personal and medical history data through a specifically designed questionnaire. Blood and urine samples were collected to find serum creatinine, plasma glucose and urine protein. Results of the study showed that the overall prevalence of CKD in the Screening and Early Evaluation of Kidney Disease Project(SEEK)-India cohort was 17.2% with a mean eGFR of 84.27 ± 76.46 versus 116.94 ± 44.65 mL/min/1.73 m^2 in non-CKD group while 79.5% in the CKD group had proteinuria. Prevalence of CKD stages 1, 2, 3, 4 and 5 was 7%, 4.3%, 4.3%, 0.8% and 0.8%, respectively. They also reported the most common risk factors and other characteristics among the subjects diagnosed with CKD as hypertension (64.5%), anemia (40.7%) and diabetes (31.6%).

Ahmed I et al, 2006[49] conducted a study to estimate the prevalence of proteinuria in a rural adult population in Vellore district, Tamil Nadu. A convenient sample of 5043 adults was included. All individuals were tested for albuminuria by albumin dipstick examination in an untimed urine sample. Individuals who tested positive for albuminuria underwent a second dipstick examination after a gap of one week. Individuals with persistent albuminuria on the second dipstick examination underwent further evaluation which included medical history, physical examination, 24 h urine protein estimation, total serum protein and albumin estimation. Ultrasound of the abdomen was done in patients with renal failure and renal biopsy was performed in selected patients. Of the total 5043 individuals screened, 63.1 per cent were females. Mean age of the study population was 50.94 ± 11.2 yr. First dipstick test identified 594 individuals positive for albuminuria. Repeat dipstick could be done in only 576, of whom 212 showed persistent albuminuria. Significant proteinuria was

detected in 24 individuals of the 208 who had 24 h urine protein measured. Of these 24 patients, 3 were found to have chronic renal failure, 12 were presumed to have diabetic nephropathy clinically, one each had focal segmental glomerulosclerosis. The prevalence of proteinuria in this adult rural population was 0.47 per cent (0.30-0.67%). The detection and treatment of chronic kidney disease in 24 individuals is bound to reduce the rate of decline of renal functions. Screening programme for proteinuria in different parts of country may be an effective measure to bring a decline in rate of progression of chronic kidney disease in general population.

Etiological Factors in Chronic Kidney Disease

Salman M et al, 2015[50]conducted a study to evaluate the attributable causes of CKD in an adult population at a tertiary referral hospital. It was a retrospective study at Hospital Universiti Sains Malaysia (HUSM). This was an analysis based on medical records of adult patients at HUSM. Data regarding demographics, laboratory investigations, attributable causes and CKD stage were gathered. A total of 851 eligible cases were included. The patients' mean age was 61.18 ± 13.37 years. CKD stage V was found in 333 cases (39.1%) whereas stages IV, IIIb, IIIa, and II were seen in 240 (28.2%), 186 (21.9%), 74 (8.7%) and 18 (2.1%), respectively. The percentage of CKD stage V patients receiving renal replacement therapy was 15.6%. The foremost attributable causes of CKD were diabetic nephropathy (44.9%), hypertension (24.2%) and obstructive uropathy (9.2%). The difference in the prevalence of CKD due to Diabetic nephropathy, hypertension and glomerulonephritis between patients ≤50 and >50 years old was statistically significant. The study results suggested that DN and hypertension are the major attributable causes of CKD among patients at a Malaysian tertiary-care hospital.

Tuttle KR et al, 2014[51] reported in a consensus report that diabetes as a major cause for chronic kidney disease. The incidence and prevalence of diabetes mellitus have grown significantly throughout the world, due primarily to the increase in type 2 diabetes. This overall increase in the number of people with diabetes has had a major impact on development of diabetic kidney disease, one of the most frequent complications of both types of diabetes. Diabetic kidney disease is the leading cause of end-stage renal disease (ESRD), accounting for approximately 50% of cases in the developed world.

Tzanakaki E et al, 2014[52] examined the causes and complications of chronic kidney disease in patients on dialysis. The study population consisted of all prevalent patients on peritoneal dialysis (PD) or haemodialysis in the University Hospital of Heraklion, Crete. Causes of morbidity and hospitalizations were examined for all dialysis patients with at least one admission in the renal ward. Mortality was examined for a period of 33 months. An overall of 123 patients with diagnosed renal failure were studied. The data were collected through the patients medical records. Out of the 123 patients with ESRD who participated in the study, 55.3% were men with a mean age of 65.3±15.2 years (range 16-85 years). The majority of patients (62.6%) were on HD, while 37.4% were on PD. Results showed that the major cause which seems to be responsible for the occurrence of chronic kidney disease as diabetic kidney disease (19,5%), followed by glomerulonephritis (18,7%). The major causes of hospitalization were infections (37.9%), including bacteraemia due to central catheter infection (40.4%), peritonitis in PD patients (19.1%), gastroenteritis (12.8%), respiratory tract infections (12.8%), urinary tract infections (6.4%) and other infections (such as cholangitis, skin infections etc) 8.5%.

Cardiac problems as a reason for hospitalization included pulmonary edema (57.1%), faint episodes, pulmonary embolism, decompensated heart failure and myocardial infarction (7.1% each). The study results concluded that the two major causes of hospitalization emerged in this study: catheter related infections and pulmonary edema.

Nand N et al, 2012[53] conducted a study to evaluate the risk factors and their degree of reversibility in cases of acute and on chronic renal failure admitted to a tertiary care hospital over a period of one year. In this study 100 patients of acute-on-chronic renal failure (rise in serum creatinine of 0.5 mg/dl, if baseline serum creatinine was < 3 mg/dl or rise of 1 mg/dl, if baseline was > 3 mg/dl within a one-week period) were included and various reversible risk factors and the degree of reversibility of renal failure was determined. A thorough clinical evaluation and investigations of all patients was done and they were put on conservative management along with specific management of reversible factors and haemodialysis, wherever needed. The observations of various parameters were recorded at baseline and subsequently at 1 week and 2 week periods which included 24-hour urine volume, blood urea, serum creatinine, and creatinine clearance. Reversibility of these parameters was then statistically analysed. To compare the degree of reversibility, the patients were divided into 4 groups at the time of admission depending upon their GFR; group 3 with GFR 30 - 59 ml/min, group 4 with GFR 15 - 29 ml/min, group 5a with GFR 5 - 15 ml/min, and group 5b with GFR < 5 ml/min, respectively. Majority of patients were found to have more than one reversible risk factor. These were hyperuricaemia (89), electrolyte imbalance (51), infection/sepsis (47), accelerated hypertension (21), volume depletion (18),

urinary tract obstruction (16), and hypotension (7). A considerable degree of reversibility was detected, maximum being in volume depletion and urinary tract obstruction. Therefore it was concluded that patients presenting in a severe uremic state may not be suffering from ESRD and each patient should be investigated for the presence of reversible risk factors so that renal function can be restored and hence the need of renal replacement therapy can be delayed.

Venkatachalam J et al, 2012[54] conducted a study to assess the risk factors of CKD. A community based cross sectional study was conducted among the 1200 respondents, 20 years and above, residing in a coastal area of Villupuram district, over a period of 6 months using a pre-structured and validated questionnaire. The questionnaire included information on demographic characteristics, lifestyle habits, and awareness of CKD risk factors. Random blood sugar and spot urinary protein and sugar using urinalysis dipsticks were measured. Results showed that majority of the respondents were females (67.8%) with maximum belonging to the age group of 20-29yrs (23.8%), followed by 30-39yrs (22.8%). Most of them belonged to upper lower class (31.8%), followed by lower middle class (25.8%) of which 93.1% of the population were Muslims with 87.9% belonging to backward caste. The prevalence of CKD risk factors among the respondents were Proteinuria (12.5%), Glycosuria (8.4%), Hypertension (24.3%), Diabetes Mellitus (18%) and Obesity (44.4%).

Fisher MA et al, 2008[55] conducted a study to investigate the role of periodontal disease and other non-traditional risk factors in patients with CKD by: (1) describing and quantifying the relationship between CKD and periodontal disease, along with the relationship between CKD and other non-traditional and traditional risk factors, in the Third National Health and Nutrition Examination

Survey (NHANES III); and (2) developing a multivariable model for the association between CKD and periodontal disease and other non-traditional risk factors, adjusting for traditional risk factors to identify risk factors independently associated with the presence of CKD. Data used in this study are: (1) responses to questions regarding age, race/ethnicity, gender, income, education, smoking, diabetes, and hypertension; (2) laboratory assays of serum cotinine, glycated hemoglobin, plasma glucose, serum CRP, serum creatinine, serum total cholesterol, serum HDL cholesterol, serum LDL cholesterol, urinary albumin, and urinary creatinine; and (3) clinical examination data for systolic and diastolic blood pressure, height, weight, and periodontal status. Chronic kidney disease prevalence was 3.6%; periodontal disease prevalence was 6.0%; and edentulism prevalence was 10.5%. Adults with periodontal disease and edentulous adults were twice as likely to have chronic kidney disease (adjusted odds ratio, 1.60; 95% confidence interval, 1.16 to 2.21; adjusted odds ratio, 1.85; 95% confidence interval, 1.34 to 2.56, respectively) after simultaneously adjusting for other traditional and non traditional risk factors.

Middleton RJ et al, 2006[56] did a study to estimate the burden of CKD in patients with diabetes and to examine the ability of serum creatinine and albuminuria to detect clinically meaningful CKD compared with eGFR. All adults known to have diabetes in primary and secondary care in Salford, UK, alive with independent renal function on 1 January 2004 were included in this observational study (n=7596). Demographic and laboratory parameters were obtained from the Electronic Patient Record. eGFR was determined using the 4-variable modification of diet in renal disease (MDRD) formula. Creatinine and albuminuria were measured in the preceding 2 years in 82.3 and 55.2% of subjects, respectively. In

patients with CKD, normoalbuminuria was present in 48.8%, and serum creatinine was normal in 54.7%. An abnormal serum creatinine had a sensitivity and specificity of 45.3 and 100%, respectively, to identify CKD. The combination of abnormal creatinine and albuminuria had an improved performance but still failed to detect a large number with CKD (sensitivity 82.4%, specificity 75.4%). Serum creatinine failed to identify CKD more often in females (OR 8.22, CI 6.56–10.29). The study concluded that undiagnosed CKD is common in diabetes. Current screening strategies, based on creatinine or albuminuria, fail to identify a considerable number of subjects with CKD. Incorporating eGFR into screening for CKD would identify individuals earlier in the natural history of the disease and enable early effective treatment to delay progression of CKD.

Treatments in Chronic Kidney Disease patients

Molina P et al, 2013[57] conducted a study to evaluate whether vitamin D supplementation with daily cholecalciferol could reduce albuminuria in proteinuric chronic kidney disease (CKD) patients. This 6-month prospective, controlled, intervention study enrolled 101 non-dialysis CKD patients with albuminuria. Patients with low 25(OH) vitamin D [25(OH)D] and high parathyroid hormone (PTH) levels (n = 50; 49%) received oral cholecalciferol (666 IU/day), whereas those without hyperparathyroidism (n = 51; 51%), independent of their vitamin D status, did not receive any cholecalciferol, and were considered as the control group. Results showed that cholecalciferol administration led to a rise in mean 25(OH)D levels by 53.0 ± 41.6%. Urinary albumin-to creatinine ratio (uACR) decreased from (geometric mean with 95% confidence interval) 284 (189-425) to 167 mg/g (105-266) at 6 months ($P < 0.001$) in the cholecalciferol group, and there was no change

in the control group. Reduction in a uACR was observed in the absence of significant changes in other factors, which could affect proteinuria, like weight, blood pressure (BP) levels or antihypertensive treatment. Six-month changes in 25(OH)D levels were significantly and inversely associated with that in the uACR, after adjustment by age, sex, body mass index, BP, GFR and antiproteinuric treatment. The mean PTH decreased by −13.8 ± 20.3% only in treated patients, with a mild rise in phosphate and calcium–phosphate product. The study concluded that In addition to improving hyperparathyroidism, vitamin D supplementation with daily cholecalciferol had a beneficial effect in decreasing albuminuria with potential effects on delaying the progression of CKD.

Al-Ageel NA et al, 2012[58] conducted a study to investigate current practice of anemia management in haemodialysis patients and to assess the appropriateness of anemia management by comparing observed practice to the Kidney Disease Outcomes Quality Initiative (KDOQI) guideline recommendations. Data on anemia parameters (Serum Ferritin and Transferrin saturation), comorbidities, erythropoiesis-stimulating agents (ESA) dosing and iron supplementation were collected. The data were collected for 7 months retrospectively from April to the end of May 2008 and prospectively from June to October 2008. Patients who were over 18 years of age with ESRD undergoing haemodialysis were included. Patients were excluded if they have cancer or receiving chemotherapy or radiotherapy. Data were collected from 87 patients. Mean Haemoglobin value for those patients was 11.16±0.97g/dL. Thirty-nine patients (45%) had mean Haemoglobin values between 11.0 and 12.0 g/dL, the target range recommended by KDOQI guideline. The mean weekly prescribed dose of erythropoietin was 8099±5946 IU/Week (135±99

IU/kg/Week). Information on ferritin concentrations was available for 48 (55%) patients. The mean serum ferritin concentration for those patients was 693±420.5 ng/mL. Fifty-two patients had transferrin saturation (TSAT) values recorded. The mean TSAT value was 38.5±19.7%. The study results concluded that there is an opportunity to improve anemia management in haemodialysis patients particularly thorough evaluation of causes of inadequate response rate and better monitoring and management of iron status.

Link between Chronic Kidney Disease and Oral health status

Ruokonen H et al, 2016[59] investigated whether oral disease inflammatory burden or different etiology (diabetes nephropathy vs. other etiologies) of CKD could be associated with mortality. This prospective cohort study comprised 144 adults at the predialysis stage. Clinical oral and radiologic examination was made from 2000 to 2005. Patients were followed up until August 2015. Cause of death could be verified from 62 of 65 patients. Clinical health data were combined with mortality records obtained from the Finland national statistics database. Number of teeth, total dental index (TDI), and periodontal inflammatory burden index were calculated to describe degree of oral inflammation. Results showed that the primary causes of death were cardiovascular diseases, infection, and cancer. There was a statistically significant difference in survival between diabetic nephropathy (23.8%) and other patients with CKD (59.9%; log-rank test P <0.001). Also showed fewer teeth, higher age, and diabetes mellitus were statistically significant independent risk factors for death. Deceased patients had fewer teeth (P <0.001) and higher TDI (P <0.05). Study results concluded that risk of death was higher among patients with diabetic nephropathy. The deceased had fewer teeth and more oral infections.

Chhokra M et al, 2013[60] conducted a study to evaluate the periodontal health status of ESRD patients undergoing haemodialysis and establishing the underlying association between renal failure and periodontal disease. Eighty control and test subjects were included in the study, after matching age and sex. Creatinine and GFR were measured in each patient. Oral hygiene index- Simplified (OHI-S), GI, PI, CAL were recorded as periodontal parameters to assess the correlation between the subjects of the two groups. Further, the test group was divided into three sub-groups, on basis of duration, as less than 6 months, from 6 months to one year and more than one year. Results showed that statistical significant difference was observed for all periodontal parameters between the test and control group. However, difference amongst periodontal parameters on basis of duration of haemodialysis was seen between the subgroups of test subjects, it was not found to be statistically significant. They concluded that the severity of periodontal diseases in ESRD patients undergoing haemodialysis was majorly affected due to debilitating condition of the subjects.

Brito F et al, 2012[19] in a cross-sectional study determined the extent and severity of periodontitis in chronic kidney disease patients undergoing the following three different treatment modalities: predialysis; continuous ambulatory peritoneal dialysis (CAPD); and HD; and then they compared the findings with those from systemically healthy individuals. Forty CAPD patients, 40 HD patients, 51 predialysis patients and 67 healthy individuals were examined. The periodontal examination included probing pocket depth, clinical attachment loss, bleeding on probing and presence of plaque. Patients with at least four sites with clinical attachment loss $\geq$ 6 mm were considered to have severe chronic periodontitis, and

those with at least 30% of sites with clinical attachment loss ≥ 4 mm were considered to have generalized chronic periodontitis. Study results showed that predialysis and HD patients had significantly more sites with clinical attachment loss ≥ 6 mm than healthy individuals. The CAPD patients had similar periodontal condition to healthy subjects and there were significantly more cases of severe chronic periodontitis in predialysis and HD patients. They concluded that predialysis and HD are associated with a higher prevalence of severe periodontitis compared with healthy individuals and CAPD patients.

Cengiz MI et al, 2009[61] conducted a study to investigate the dental and periodontal health status of patients on regular HD maintenance therapy, and the effect of duration of HD on oral status. Sixty-eight HD patients and 41 controls were examined. Decayed, Missing or Filled Teeth (DMFT index), PI, GI, PPD and loss of periodontal attachment (LPA) were examined. Except DMFT index, significant differences were found in the other index values between patients and controls. Dialysis duration significantly correlated not with DMFT but with the others. Also, DMFT values showed no significant differences between the five HD subgroups. For the PI, GI and PPD values, the first 5-year period revealed no significant variation, whereas the second 5-year period included significant increases. After 10 years, a much more significant increase was observed. The LPA values did not show any significant differences between the HD subgroups, but after 10 years a significant progressive increase can be observed. They concluded that the dental and periodontal health is poor in HD patients and becomes worse with time on dialysis. Thus, oral health maintenance is of utmost importance in this patient group.

Chamani G et al, 2009[62] did a cross-sectional study on two groups of patients: one including 68 renal dialysis patients (test) and the other including 30 healthy subjects (control). Half-mouth measurements of GI, PI, PPD, gingival recession (GR), CAL, BOP as well as DMFT index were recorded. The study results showed that the GI, BOP, PPD, CAL and GR were significantly greater among the test group as compared with the control group; however, the DMFT did not differ significantly among the groups. There was no relationship between the duration of the dialysis and the periodontal indices. They concluded that the patients with chronic renal failure have less favourable periodontal health than normal patients.

Joseph R et al, 2009[63] assessed the prevalence of periodontal disease among a group of patients with renal disease and compared their periodontal status to that of healthy controls. 77 patients with different forms of renal disease and 77 healthy controls were examined for oral hygiene status, gingival inflammation, PPD and CAL. The subjects were grouped into three as no/mild, moderate and severe periodontitis. Results showed that all periodontal parameters were significantly elevated in the case group as compared to controls ($p < 0.001$). The prevalence and severity of periodontal disease was also significantly higher in the case group ($p < 0.001$). This study provided evidence for a greater prevalence and severity of periodontal disease among patients with renal disease.

Yoshihara A et al, 2007[64] conducted a study to investigate is a link exists between periodontal disease and chronic renal function in community dwelling elderly subjects. A total of 145 study subjects, all 77 years of age, participated in this study. A periodontal examination was carried out by trained dentists. Urine was collected over 24 hours, and blood was taken on the morning of the dental exam.

The volume of creatinine per 24 hours and volume of urine per 24 hours were used as urinary markers of kidney function; serum creatinine levels (Cre-S) were used as a blood marker of kidney function. Creatinine clearance per 24 hours was calculated as Cre-U/Cre-S. In addition, biochemical parameters of bone turnover were measured: urinary deoxypyridinoline (U-DPD) as a bone resorption marker and serum osteocalcin (S-OC) as a bone formation marker. Multiple regression analysis was used to evaluate the relationship between the percentage of periodontal sites with ≥6-mm clinical attachment level (% ≥6-mm CAL) and renal function, as well as the relationship between % ≥6-mm CAL and bone metabolism. The % ≥6-mm CAL was used as the dependent variable. Multiple regression analysis showed that creatinine clearance per 24 hours and S-OC were significantly associated with % ≥6-mm CAL per person. The standardized coefficients were 0.26 ($p = 0.015$) and -0.27 ($p = 0.006$), respectively. Study concluded that the % ≥6-mm CAL was significantly associated with renal function and bone metabolism markers. This study suggests that the increased incidence of chronic renal failure that occurs with age might increase the probability of severe periodontal disease in community-dwelling elderly subjects.

Davidovich E et al, 2005[65] did a study to describe the oral condition of chronic renal failure and healthy subjects, and the relationship between oral variables, chronic renal failure (CRF) conditions, and their treatment. Four renal failure groups were included. They were: chronic renal disease (n=22); undergoing dialysis (n=22); after dialysis and transplant (n=21); and after transplant (n=32), and a healthy control (n=38) were examined. Caries, enamel hypoplasia, pulp obliteration, PI, gingival bleeding, recession, overgrowth and index, probing depths,

attachment loss, renal treatments and their relations with the oral variables were analysed. Results showed that the renal failure groups had higher GI and bleeding, probing depths, attachment loss, hypoplasia and obliteration and less caries, than the control. Plaque was higher in the dialysis and pre-dialysis groups. Overgrowth was evident after transplant. The pre-dialysis group showed lower GI than other renal groups. Dialysis duration and end-stage renal failure significantly correlated with gingivitis, probing depth, attachment loss and enamel hypoplasia. Immuran correlated positively with probing depth, gingival recession and attachment loss. Normiten and Nifedipine had positive correlations with gingival overgrowth. This study concluded that CRF patients are characterized by pulp obliteration, gingival and periodontal diseases and also that the duration of end stage renal failure and type of systemic treatment have a significant influence on the oral condition.

Klassen JT et al, 2002[24] conducted a study to find the dental health status of dialysis patients. Completion of a questionnaire and a non-invasive oral examination was obtained from haemodialysis and peritoneal dialysis patients registered in the dialysis program at St. Paul's Hospital in Saskatoon, Saskatchewan, as of March 1, 1999. Information was also gathered from the medical chart. Medication history, history of diabetes, hypertension, and nondental prosthetic devices were also recorded. Of the 226 dialysis patients in central and northern Saskatchewan, 147 were interviewed and examined. Of these, 94 (64%) were dentate, and the same number had been on dialysis for a mean of more than 2 years; about a third were diabetic, almost all were hypertensive and all had non-dental prosthetic devices or arteriovenous fistulae, or both. Sixty (64%) of the dentate patients were candidates for kidney transplantation. Most of the dentate patients reported brushing once or

more daily, but they flossed infrequently or never. Dental visits were infrequent, less than every 5 years in 59 (63%) of the dentate patients. Findings in the dentate group included increased tooth mobility, fractures, erosion, attrition, recession, gingivitis and a high plaque index.

Effect of periodontal disease on renal disease parameters

Grubbs V et al, 2016[66] investigated if periodontal disease brings about kidney function decline over time. In a longitudinal retrospective cohort of 761 elderly men with preserved kidney function [estimated GFR > 60 mL/min/1.73 m^2 using a calibrated creatinine and cystatin C (eGFRcr-cys) equation] at baseline, multivariable Poisson's regression was performed to examine the association of severe periodontal disease with incident CKD, defined as incident eGFRcr-cys<60 mL/min/1.73 m^2 and rapid (>5% annualized) eGFRcr-cys decline. At baseline, the mean age was 73.4 (SD 4.8) years, the median eGFRcr-cys was 82.4 mL/min/1.73 m2, and 35.5 and 25.4% of participants had severe periodontal disease by European Workshop and CDC (Centre for Disease Control and Prevention)/AAP (American Academy of Periodontology) criteria, respectively. After a mean follow-up of 4.9 years (SD 0.3), 56 (7.4%) participants had incident CKD. Severe periodontal disease was associated with a 2-fold greater rate of incident CKD after adjusting for confounders compared with not severe periodontal disease by European Workshop criteria but did not reach statistical significance by CDC/AAP criteria. They concluded that severe periodontal disease may be associated with incident clinically significant kidney function decline among a cohort of elderly men.

Han SS et al, 2013[40] conducted a study to examine the correlations between periodontitis and both decreased GFR and urinary abnormalities, including

proteinuria and haematuria. Data on 15,729 Korean adults were obtained from the Korean National Health and Nutritional Examination Surveys IV and V. The CKD markers included a decreased estimated GFR (eGFR;<60 mL/min/1.73 m(2)), proteinuria, and haematuria. Odds ratios (ORs) and 95% confidence intervals were measured using stepwise multivariate logistic regression analyses for CKD markers based on the presence of periodontitis. Patients with periodontitis had greater unadjusted ORs for CKD markers compared to those without periodontitis, as follows: decreased eGFR, 4.07 (3.11-5.33); proteinuria, 2.12 (1.48-3.05); and haematuria, 1.25 (1.13-1.39, all P<0.001). Periodontitis was a significant predictor of decreased eGFR independent of all covariates. However, the effect of periodontitis on decreased eGFR seemed to be affected by hypertension and diabetes mellitus. Periodontitis was not an independent predictor of proteinuria; the significance disappeared after adjusting for hypertension and diabetes mellitus. Periodontitis was significantly correlated with haematuria, leading to similar ORs regardless of the adjustment for covariates. This study confirmed the correlation between periodontitis and CKD markers, including decreased eGFR, proteinuria, and haematuria in Korean adults.

Kshirsagar AVet al, 2007[67] examined relationship between periodontitis and two measures of systemic inflammation, serum albumin and CRP were examined among patients who were receiving chronic outpatient haemodialysis. Adult patients at two locations, North Carolina and New York City were evaluated by dentist examiners. Multivariable logistic regression was used to determine the association of periodontitis with low serum albumin and with high CRP. Total 154 patients completed the study. Study results showed that severe periodontitis was

associated with low serum albumin compared with individuals without severe periodontitis disease after adjustment for age, gender, race, diabetes, hypertension, body mass index, smoking, study site, total cholesterol, serum calcium, serum phosphorus, normalized protein catabolic rate. It also showed that there was no observed association of severe periodontitis with CRP.

Bots CP et al, 2006[68] in a study aimed to compare the oral health status of chronic renal failure patients on renal replacement therapy with a matched reference population. This was a cross-sectional study with 42 dentate CRF patients - aged 25-52 years old and were matched with a reference group of 808 dentate subjects. The oral health was assessed using decayed missing filled (DMF) indices, simplified oral hygiene index and periodontal status. An oral health questionnaire was used to assess self-reported dental problems. Student t-tests and chi-square tests were performed to compare the chronic renal failure patients with the controls. All index-scores in the CRF patients were comparable with the controls except for number of teeth covered with calculus that was significantly higher ($P < 0.05$) in chronic renal failure patients (4.1 ± 2.6) than in controls (3.0 ± 2.9). The self-reported oral health questionnaire revealed a trend for increased temporomandibular complaints in chronic renal failure patients (16.7% vs 5.7% in controls; P ¼ 0.06) as well as bad taste (31.0% vs 6.8% in controls,P ¼ 0.08). They concluded that for most dental aspects, the oral health of chronic renal failure patients is comparable with controls.

Effect of periodontal treatment on renal disease parameters

Fang F et al, 2015[69] conducted a study to evaluate the effects of non-surgical periodontal therapy on the clinical response and systemic status of ESRD patients. Patients in the intervention group (n = 48) received nonsurgical periodontal therapy

and then a supragingival prophylaxis at the 3-month follow up, and those in the control group (n = 49) received no intervention throughout the study. At 6 weeks, 3 months, and 6 months after therapy, clinical periodontal examinations were conducted and blood samples were taken to evaluate inflammatory, nutritional and lipid profiles. The results showed a significant improvement in clinical periodontal parameters in the intervention group. Compared to the control group, the intervention group had significantly lower high-sensitivity C-reactive protein at 3 months and 6 months. Significant improvements were found in interleukin-6, ferritin, albumin, creatinine, blood urea nitrogen, and transferrin after therapy. The study results concluded that non-surgical periodontal therapy can effectively improve periodontal, circulating inflammatory and nutritional status in ESRD patients. Non-surgical periodontal therapy, as a relatively simple intervention, has beneficial systemic effects in ESRD patients.

Chakraborty S et al, 2014[70] conducted a study to investigate differences in concentrations of serum ferritin in patients with and without periodontal disease before and after non-surgical periodontal therapy and correlate these values with clinical variables associated with periodontal disease. Forty-two individuals were included in this study, 20 with chronic periodontitis and 22 classified as periodontally healthy. Serum ferritin concentrations, haemoglobin levels, and periodontal parameters (PD, CAL, GI, BOP and PI) were recorded at baseline and 3 months after non-surgical periodontal therapy. Results of the study showed that patients with CP showed higher concentrations of serum ferritin than periodontally healthy controls. Also a positive and significant correlation was observed between serum ferritin levels and the number of sites with PD ≥6 mm at baseline. Significant

reductions in serum ferritin levels were observed at the 3-month assessment after periodontal treatment, and the post-treatment serum ferritin values were comparable to those of controls (P >0.05). Furthermore, the post-treatment degree of change in the serum ferritin level was positively and significantly associated with improvement in PD. Study results concluded that serum ferritin levels are raised in patients with CP and decrease to control levels post-treatment.

de Souza CM et al, 2014[71] investigated the impact of oral health indicators, chronic periodontitis and its treatment on survival rates in a group of patients undergoing haemodialysis. Clinically stable patients undergoing HD were referred for a dental examination. All patients were prospectively followed in the dialysis clinic, and all-cause mortality was recorded. Three groups of patients were analyzed: those who received CP treatment, those who did not, and patients without CP as a control group. A total of 122 patients were enrolled. Results showed that forty percent reported having rarely been evaluated by a dentist, and 59% had CP. There were 34 fatal events during a mean follow-up time of 64.1 ± 11.2 months. Oral factors associated with death in the univariate analysis were decreased frequency of dental visits; non-use of dental floss; increased decayed, missing, and filled teeth index; presence of CP; and absence of CP treatment. Patients with CP had a higher risk of death from all causes compared with patients without CP in the univariate analysis for untreated patients and to a lesser extent for treated patients. These significant differences were not maintained after adjustments for confounders in the multivariate model. The study results concluded that poor oral health, including CP, is a common finding in patients undergoing HD.

Siribamrungwong M et al, 2013[72] conducted a study to evaluate the association between chronic systemic inflammation and periodontal status and the effect of periodontal treatment in peritoneal dialysis patients. Clinical periodontal status was evaluated in 32 stable chronic peritoneal dialysis patients by PI and periodontal disease index. Hematologic, chemical, nutritional, and dialysis-related data as well as highly sensitive C-reactive protein were analyzed before and after periodontal treatment. At baseline, high sensitive CRP positively correlated with the clinical periodontal status (plaque index; r = 0.57, $P < 0.01$, periodontal disease index; r = 0.56, $P < 0.01$). After completion of periodontal therapy, clinical periodontal indexes were significantly lower and high sensitivity CRP significantly decreased from 2.93 to 2.21 mg/L. Moreover, blood urea nitrogen increased from 47.33 to 51.8 mg/dL, reflecting nutritional status improvement. Erythropoietin dosage requirement decreased from 8000 to 6000 units/week while haemoglobin level was stable. Periodontitis is an important source of chronic systemic inflammation in peritoneal dialysis patients. The study results concluded that treatment of periodontal diseases can improve systemic inflammation, nutritional status and erythropoietin responsiveness in peritoneal dialysis patients.

Wehmeyer MMH et al, 2013[73] conducted a study to determine the impact of treatment on clinical measures of periodontitis severity on dialysis patients; (2) to determine the impact of periodontal treatment on pre-specified laboratory parameters (serum albumin and interleukin-6); and (3) to assess recruitment and retention of patients. Dialysis patients with moderate or severe CP were selected. A total of 342 dialysis patients were approached for participation: 53 were randomized, with 26 participants assigned to immediate treatment and 27 to a control arm for

treatment after 6 months. Fifty one patients completed baseline appointments; 46 were available for 3 month follow up and 45 were available for 6 month follow up examinations. Forty three participants completed all visits. At 3 months, there was a statistically significant improvement for the treatment group compared to the control group for 3 periodontal parameters: mean PD (p=0.008), extent PD ≥ 4 mm (p=0.02), and extent GI ≥1 (p=0.01). By 6 months, however, the difference between groups was no longer present for any variable except PD ≥ 4 mm (p=0.04). There was no significant difference between the groups for serum albumin or high-sensitivity interleukin 6 at any time point, when adjusted for BMI, diabetic status, and plaque index.

Yazdi FK et al, 2013[74] conducted a study to evaluate the impact of nonsurgical periodontal treatment on the serum levels of CRP in CKD patients on haemodialysis. A total of 77 CKD patients on haemodialysis were included in this study. At baseline, periodontal examination was assessed for all the patients, and chronic periodontitis was defined through CAL and PPD, according to the AAP. Nonsurgical periodontal treatment was performed and serum levels of CRP were evaluated at baseline and 8 weeks after periodontal treatment. Periodontal treatment resulted in significant reductions in CRP levels. The difference between pre and post-treatment CRP concentrations did not show any significant relationship with the severity of periodontitis. Study results concluded that nonsurgical periodontal treatment can effectively reduce the serum level of CRP in these patients.

Ardalan M R et al, 2011[75] in a study assessed the severity and possible role of periodontitis in a group of patients with unknown primary glomerulonephritis. The study included ten patients with unknown primary glomerulonephritis, and who had a

renal biopsy with stable renal function and serum creatinine <1.6 mg/dL, were recruited. Severity of the periodontal disease was clinically measured with PI, GI and PPD. The dental treatments were aimed to eradicate the inflammatory/infectious diseases from the oral cavity by a wide variety of treatments, including oral hygiene instruction, nonsurgical or surgical periodontal therapy, systemic or topical medicaments, root canal therapy, and extraction, if needed. The patients were also put on angiotensin-converting enzyme inhibitor or angiotensin receptor blockers for controlling blood pressure and proteinuria. Six months following appropriate periodontal treatment, renal function, degree of proteinuria, and level of CRP were measured in each individual. The study results showed that the median urine protein excretion was lower following the periodontal therapy. Prior to the dental and/or periodontal therapies, the median PI, PD, and GI were 57.5%, 4.3, and 1.1, respectively. The majority of the patients had advanced periodontal disease. In four patients, +2/+3 CRP turned negative after periodontal treatment. This study revealed that a causative link might exist between periodontal disease and glomerulonephritis.

Vilela EMet al, 2011[32] conducted a study to determine the impact of periodontal treatment on serum levels of prohepcidin (the prohormone of hepcidin) and systemic inflammation markers, as well as correlations among these markers, in patients with chronic periodontitis and chronic kidney disease who were not undergoing dialysis. This study consisted of 56 CP patients, 36 with chronic kidney disease and 20 without systemic diseases and with normal renal function (control group). The inflammatory markers ultrasensitive C-reactive protein, interleukin-6, and prohepcidin were evaluated before and 3 months after periodontal treatment. The following blood parameters were also found: complete hemogram (automated

Coulter STKS), serum iron (ferrozine), ferritin (electrochemiluminescence), and transferrin saturation index (Labtest ferrozine). The efficacy of periodontal treatment was confirmed by the improvement in clinical parameters of chronic periodontitis in the control and chronic kidney disease groups. Periodontal treatment resulted in significant reductions in ultrasensitive C-reactive protein, interleukin-6 and serum prohepcidin levels in both groups. There was also a reduction in prohepcidin after periodontal treatment was significantly and independently associated with interleukin-6 levels in the control group. They concluded that by inducing a decline in the systemic inflammatory response and a decrease in serum prohepcidin, successful periodontal treatment may represent an important means of ameliorating the inflammatory burden seen in patients with chronic kidney disease.

Artese et al, 2010[76] conducted a study to investigate how predialysis CKD patients with periodontitis respond to non-surgical periodontal treatment. Twenty-one predialysis patients (group 1) and 19 individuals without clinical evidence of kidney disease (group 2) with chronic periodontitis were subjected to non-surgical periodontal treatment with no antibiotics. Clinical periodontal and systemic parameters were evaluated at baseline and 3 months after treatment. Both groups showed significant and similar post-treatment improvements in all periodontal parameters examined. Most interestingly, periodontal treatment had a statistically significant positive effect on the glomerular filtration rate of each individual. Our results indicate that chronic periodontitis in predialysis kidney disease patients improved similarly in patients with chronic periodontitis and no history of CKD after receiving non-surgical periodontal therapy. This study demonstrates that CKD predialysis patients show a good response to non-surgical periodontal treatment.

MATERIALS & METHODS

Study design:

This is a comparative interventional study for evaluating the level of C - reactive protein levels and iron indices in chronic periodontitis patients and in chronic kidney patients undergoing haemodialysis with chronic periodontitis before and after scaling and root planing procedure.

Study settings:

The chronic periodontitis patients for the study were selected from the Outpatient of Department of Periodontics and Oral Implantology, Sree Mookambika Institute of Dental Sciences, Kulasekharam and chronic kidney disease patients undergoing haemodialysis where referred from Department of Nephrology, Sree Mookambika Institute of Medical Sciences, Kulasekharam for oral prophylaxis.

Study duration

The study duration was of 6 months, which lasted from July 2016 to December 2016.

Informed consent and Ethical considerations

The study protocol was approved by the Institutional Research Committee of Sree Mookambika Institute of Dental Sciences (Ref No. 18/06/2015, Annexure-1) and also by the Institutional Human Ethics Committee of Sree Mookambika Institute of Medical Sciences, Kulasekharam (Ref No: SMIMS/IHEC/2-15/A/05, Annexure-2) and was registered under the Clinical Trial Registry of India. The Clinical trial registration number is REF/2016/01/010407

Study protocol was explained to patient and the information on the nature and potential benefit of their participation in the study was also explained

(Annexure-3). Informed consent was obtained from all subjects after screening (Annexure-4).

Sample size calculation:

Sample size was calculated based on the previous study by Vilela EM et al, 2011[32]

In which it was reported that the C- reactive protein values as follows.

Chronic periodontitis patients with CKD = 6.18±5.39(mg/L)

Chronic periodontitis patients without CKD (controls) = 3.04±3.82(mg/L)

The sample size $n = \frac{2(z\left(1-\frac{\alpha}{2}\right)+z(1-\beta))^2}{\Delta^2}$

Where $z_{(1- a /2)}$ is the alpha error whose value for significance level of 10%, is 1.645 and $z_{(1- b)}$ is the beta error or power of the study whose value power of 80% is 0.8416.

$$\Delta = \frac{\text{Difference in means}}{\text{S.D}} = \frac{6.18 - 3.04}{3.82} = 0.822$$

Alpha error at 5% significance level = 1.645

Beta error (power) at 80 % = 0.8416

Sample size $n= \frac{2(1.645 + 0.8416)^2}{0.822^2} = \frac{12.3664}{0.6757}$ =18.3 rounded off to 20 for each group

The number of participants required in each intervention group is 20 for a Significance level of 10% and power of 80%

Total sample size = 3x 20 = 60

The selected patients were assigned into 3 groups; each group consisting of 20 patients.

Group I - Twenty Chronic kidney disease patients undergoing haemodialysis for less than a year with chronic periodontitis

Group II - Twenty Chronic kidney disease patients undergoing haemodialysis for more than a year with chronic periodontitis

Group III - Twenty systemically healthy chronic periodontitis patients

Inclusion criteria:

Group I: Twenty Chronic kidney disease patients undergoing haemodialysis for less than a year with chronic periodontitis

i. Patients with chronic periodontitis was diagnosed when there was two or more interproximal sites with CAL ≥4 mm, not on the same tooth, or two or more interproximal sites with PPD ≥5 mm, not on the same tooth (Page and Eke, 2007)[33]
ii. Patients with ≥ 20 teeth
iii. Patients with bleeding on probing.
iv. Patients undergoing haemodialysis for atleast 3months and less than a year.

Group II: Twenty Chronic kidney disease patients undergoing haemodialysis for more than a year with chronic periodontitis

i. Patients with chronic periodontitis was diagnosed when there was two or more interproximal sites with CAL ≥4 mm, not on the same tooth, or two or more interproximal sites with PPD ≥5 mm, not on the same tooth.[33]
ii. Patients with ≥ 20 teeth

iii. Patients with bleeding on probing.

iv. Patients undergoing haemodialysis for more than a year.

Group III: Twenty systemically healthy chronic periodontitis patients

i. Patients with chronic periodontitis was diagnosed when there was two or more interproximal sites with CAL ≥4 mm, not on the same tooth, or two or more interproximal sites with PPD ≥5 mm, not on the same tooth.[33]

ii. Patients with ≥ 20 teeth

iii. Patients with bleeding on probing.

Exclusion criteria:

1. Edentulous patients
2. Smokers, alcoholics, pregnant women and lactating mothers.
3. Patients who had undergone periodontal treatment in the past 6 months

Clinical parameters

Periodontal examination was conducted in the Department of Periodontics, Sree Mookambika Institute of Dental Sciences, Kulasekharam.

The following clinical parameters were assessed using William's graduated periodontal probe. Clinical parameters were assessed at six sites of all teeth (mesiobuccal, midbuccal, distobuccal, mesiolingual, midlingual and distolingual) at baseline, 4 weeks and after 3 months following non surgical periodontal therapy.

1. Plaque index (PI)[34]
2. Gingival index (GI)[35]
3. Bleeding on Probing(BOP) [36]
4. Probing pocket depth(PPD)
5. Clinical attachment Level(CAL)

Renal and haematological parameters

1. Serum C-reactive Proteins(CRP)
2. Total Iron Binding Capacity(TIBC)
3. Serum Iron
4. Transferrin saturation ratio(TSAT)
5. Serum Ferritin
6. Haemoglobin(Hb)
7. Erythrocyte Sedimentation Rate(ESR)
8. Serum albumin
9. Serum creatinine
10. Glomerular Filtration rate(GFR)

Armamentarium

The collection of blood and non surgical periodontal therapy were performed with the following requirements.(Colour Plate-1)

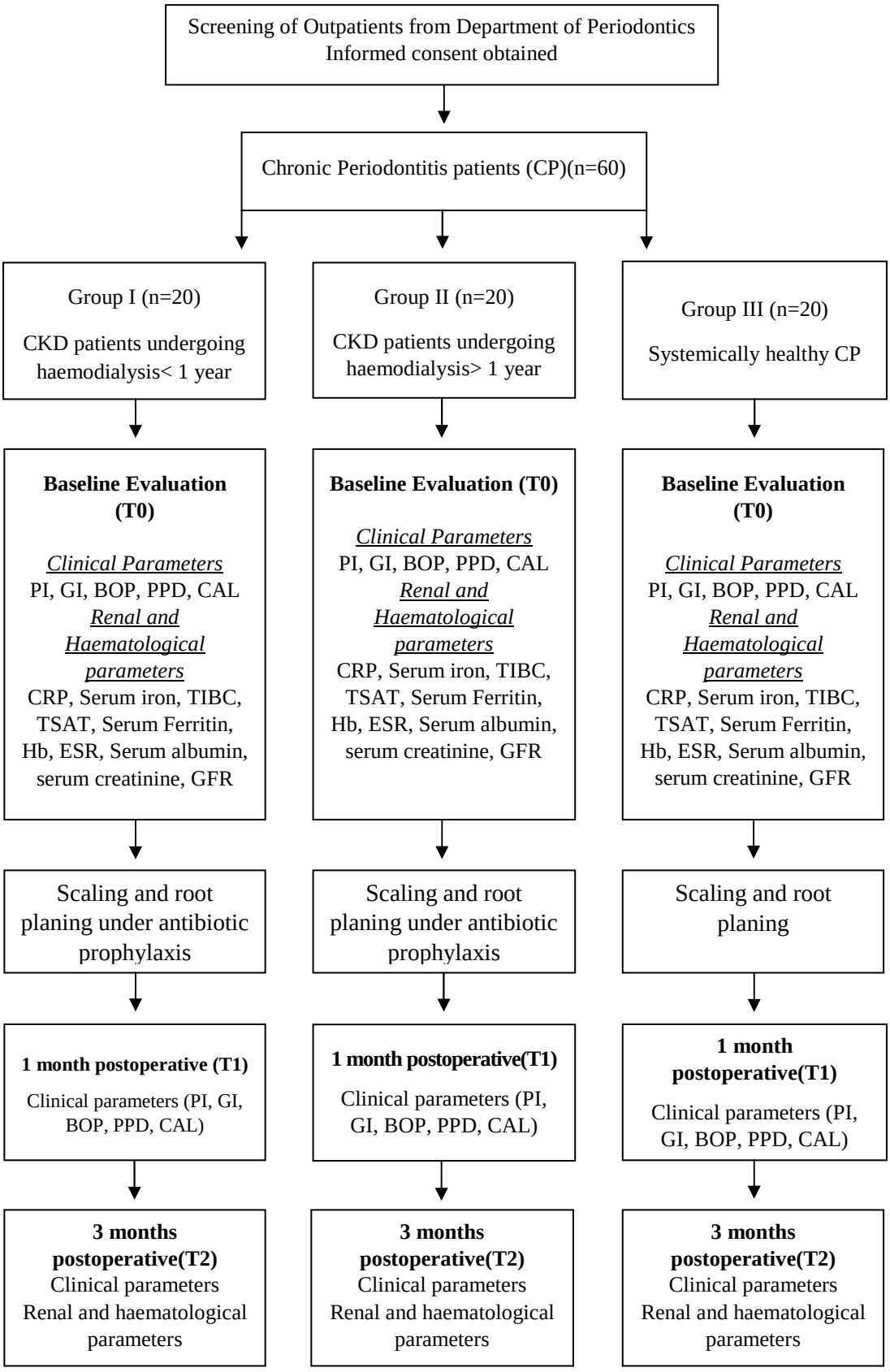
1. Mouth mirror
2. Explorer
3. William's periodontal probe.
4. Gracey Area specific curettes.
5. 23 gauge 5ml disposable plastic syringe.
6. 25 gauge 2ml disposable plastic syringe.
7. Clot activator tube.
8. KEMI C-48 centrifuge.

Procedure

After the patients were included in the study, blood samples were collected and sent to the Central laboratory of Sree Mookambika Institute of Medical Science for the estimation of renal and haematological parameters. For Group I and group II systemic antibiotics [2g amoxicillin or 600mg Clindamycin (if patient had an allergy to amoxicillin)] were administered 1 hour prior to the periodontal therapy appointment only, in order to minimize the effect of any transient bacteremia on the patient's dialysis catheter.[37,38] Antibiotics were not administered at any other appointment. PI, GI, BOP, PPD and CAL were recorded from each patient of the three groups. The clinical, renal and haematological parameters recorded at the baseline were given as T0. After baseline evaluation (T0), all the patients received nonsurgical periodontal therapy, which includes oral hygiene instructions, supragingival and subgingival scaling and root planing under local anesthesia using ultrasonic instruments and hand instruments as needed. (Annexure-5)

Patients were recalled after 1 month and 3 months during which clinical parameters were recorded as T1 and clinical, renal & haematological parameters recorded as T2 respectively (Annexure-6).

The study design flowchart is given below.



Sample Preparation

Under sterile conditions from each patient of three groups, 5ml of venous blood sample was collected at baseline and three months after the non surgical periodontal therapy from the ante-cubital fossa by venipuncture using 23 gauge needle and 5ml syringe (Colour Plate-2). Collected blood is transferred to a clot activator tube and placed for 30 minutes at room temperature and then centrifuged at 3000 rpm for 10min (Colour Plate-3). The serum obtained is then used for the estimation of renal and haematological parameters (Colour Plate-4).

Method

The evaluation of the renal and haematological parameters were done at the Central Lab of Sree Mookambika Institute of Medical Sciences, Kulasekharam.

1. Serum C reactive proteins levels: Analysed using Beckman Coulter Analyzer (Colour Plate-5)[Tris buffer (pH 7.5), (Reagents-Sodium Chloride, Polyethylene glycol 6000, anti-CRP Antibodies(Colour Plate-6)] by Turbidimetric method.
2. Serum iron, TIBC, creatinine and albumin: Analysed using Beckman Coulter AU 480 Fully Automated Analyzer(Colour Plate-7)
3. TSAT is measured as a percentage and is a medical laboratory value. It is the ratio of serum iron and total iron binding capacity.
4. The normal mean GFR for young adults is approximately 120-130mL/min/1.73m^2.[9]GFR calculated using the Modification of Diet in Renal Disease (MDRD) study equation.

   GFR (mL/min/1.73 m^2) = $175 \times (S_{cr})^{-1.154} \times (Age)^{-0.203} \times$ (0.742 if female).

   S_{cr} is serum creatinine.

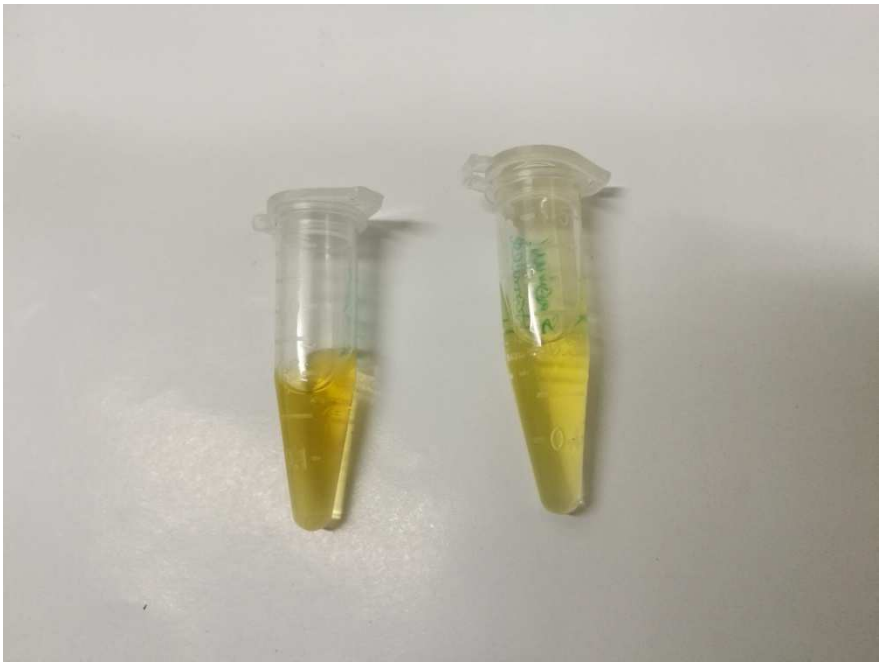
5. Serum Ferritin analysed using Beckman Coulter Access 2 Immunoassay System by Chemiluminescent detection method.(Colour Plate-8, 9, 10)
6. Hemoglobin and ESR investigations executed by automated haematology analyser and 5-part differential counter in Beckman Coulter Ac. T 5 Diff CP & Mindray BC 5300 5 diff (Method used is colorimetric method for determining Hb and Westergren method for ESR)(Colour Plate-11)

COLOR PLATES

Colour Plate-1. Armamentarium for scaling and root planing

Colour Plate-2. Blood collection

Colour Plate-3. Centrifugation



Colour Plate-4. Collected serum sample

Colour Plate -5. Autoanalyzer to measure C-reactive protein

Colour Plate -6. Reagents in the Autoanalyzer

Colour Plate -7. Beckman Coulter AU 480 Fully automated analyzer for estimation of Serum iron, TIBC, serum creatinine and albumin

Colour Plate -8. Samples placed in the sample holder for estimation of serum ferritin

Colour Plate -9. Reagents loaded in the analyzer

Colour Plate -10. Beckman Coulter Access 2
Immunoassay System for estimation of serum ferritin

Colour Plate -11. Beckman Coulter fully automated analyzer for haemoglobin estimation and ESR

RESULTS & OBSERVATIONS

The purpose of the study was to compare the effect of non-surgical periodontal therapy on periodontal parameters, renal and haematological parameters in systemically healthy chronic periodontitis patients and in chronic periodontitis patients with chronic kidney disease undergoing haemodialysis of varying duration. The study consisted on 3 groups with 20 patients in each group.

Group I : Chronic periodontitis patients with chronic kidney disease undergoing haemodialysis for less than a year

Group II : Chronic periodontitis patients with chronic kidney disease undergoing haemodialysis for more than a year

Group III : Systemically healthy chronic periodontitis patients.

Baseline data for PI, GI, BOP, PPD and CAL was recorded for all the patients in each group. Before periodontal treatment blood samples were collected and sent to the laboratory for estimation of renal and haematological parameters. After non-surgical periodontal therapy, at the end of 4 weeks and 3 months, patients were recalled and periodontal parameters were recorded. Renal and haematological parameters were recorded at the end of 3 months only.

Statistical Analysis

The data was analyzed by Statistical Package for Social Sciences (SPSS 16.0) version. The data was tested for normality. One way ANOVA (post hoc) followed by Dunnet t test was applied to find the statistical significance between the groups. Paired t test and Chi square test was applied to find the statistical significance between the T0 and T2 time within the groups. P value less than 0.05 was considered statistically significant at 95% confidence interval.

Table-1: Comparison of demographic and clinical observations between the groups

Observations	Group-I	Group-II	Group-III	p value
Age (years) (MEAN±SD)	51.15±9.66	49.90±1.20	41.35±8.79*,#	**0.04**
Gender				
Male	16	17	7*,#	**0.04**
Female	4	3	13*,#	**0.03**
BMI (kg/m^2) (MEAN±SD)	22.25±2.73	23.23±1.48	23.00±2.60	**1.34**
SBP (mmHg) (MEAN±SD)	135.80±3.66	136.50±3.03	118.40±6.03*,#	**0.04**
DBP (mmHg) (MEAN±SD)	93.80±4.25	93.30±3.06	80.40±4.08*,#	**0.04**
Number of teeth present (MEAN±SD)	27.35±3.74	27.55±3.83	28.15±3.16	**2.67**

(*p<0.05 significant compared Group-I with other groups, #P<0.05 significant compared Group-II with other groups)

Table 1 shows the demographic and the vital signs of the patients at baseline.

Table-2: Comparison of mean periodontal parameters at T0 between the groups

Groups	Plaque Index (MEAN±SD)	Gingival index (MEAN±SD)	Bleeding on probing(%) (MEAN±SD)	Probing pocket depth(mm) (MEAN±SD)	Clinical attachment level(mm) (MEAN±SD)
Group I	1.78±0.50	1.58±0.28	34.16±1.16	2.94±0.32	3.38±0.59
Group II	1.74±2.65	1.58±0.29	34.37±1.27	2.98±0.43	3.35±7.66
Group III	1.79±0.34	1.71±0.35	45.09±1.43*,#	3.10±0.52*,#	3.37±0.62
p value	**2.90**	**2.89**	**0.05**	**0.05**	**3.45**

(*p<0.05 significant compared with Group-I, #P<0.05 significant compared with)

Table 2 shows the comparison of mean periodontal parameters at baseline (T0) between the groups. This shows that there was statistically significant difference($p<0.05$) with respect to bleeding on probing and probing pocket depth in chronic periodontitis group when compared to the haemodialysis groups whereas in respect to PI, GI and CAL difference between the three groups at baseline was not statistically significant.

Table-3: Comparison of mean periodontal parameters at T1 between the groups

Groups	Plaque Index (MEAN±SD)	Gingival index (MEAN±SD)	Bleeding on probing (%) (MEAN±SD)	Probing pocket depth (mm) (MEAN±SD)	Clinical attachment level (mm) (MEAN±SD)
Group I	1.33±0.35	1.35±0.12	26.97±6.91	2.78±0.33	3.22±0.57
Group II	1.37±0.22	1.37±0.12	27.06±1.01	2.82±0.43	3.16±7.23
Group III	1.47±0.22	1.44±0.20	33.63±1.43*, #	3.00±0.45	3.26±0.57
p value	**1.67**	**1.98**	**0.05**	**2.12**	**1.45**

(*p<0.05 significant compared with Group-I, #P<0.05 significant compared with Group-II)

When comparing the mean periodontal parameters at T1 (one month postoperative) it was shown that there was no statistically significant difference in PI, GI, PPD and CAL in all the three groups, except the BOP ($p<0.05$) in group III when compared to Group I and Group II.(Table 3)

Table-4: Comparison of mean periodontal parameters at T2 between the groups

Groups	Plaque Index (MEAN±SD)	Gingival index (MEAN±SD)	Bleeding on probing(%) (MEAN±SD)	Probing pocket depth(mm) (MEAN±SD)	Clinical attachment level(mm) (MEAN±SD)
Group I	1.34±0.30	1.30±0.08	22.03±5.43	2.69±0.27	3.12±0.49
Group II	1.41±0.28	1.32±0.13	23.30±9.11	2.78±0.47	3.07±0.75
Group III	1.35±0.19	1.31±0.14	23.20±5.96	2.87±0.39	3.10±0.50
p value	**1.45**	**1.93**	**2.12**	**2.94**	**1.98**

(p>0.05 no significant difference compared between the groups)

Comparison of the mean periodontal parameters at T2 (3 months postoperative) between the groups showed that there was no statistically significant difference(Table 4).

Table-5: Comparison of mean periodontal parameters of Group-I at different time periods

Time	Group I				
	Plaque Index (MEAN±SD)	**Gingival index (MEAN±SD)**	**Bleeding on probing (%) (MEAN±SD)**	**Probing pocket depth (mm) (MEAN±SD)**	**Clinical attachment level (mm) (MEAN±SD)**
T0	1.78±0.50	1.58±0.28	34.16±1.16	2.94±0.32	3.38±0.59
T1	1.33±0.35*	1.35±0.12	26.97±6.91*	2.78±0.33	3.22±0.57
T2	1.34±0.30*	1.30±0.08	22.03±5.43*,#	2.69±0.27	3.12±0.49
p value	**0.05**	**1.56**	**0.04**	**1.34**	**2.19**

(*p<0.05 significant compared with T0, #p<0.05 significant compared with T1)

When comparing the periodontal parameters of group I at different time, it was shown that all the periodontal parameters improved from T0 to T2, but the decrease was statistically significant ($p<0.05$) only in relation to PI and BOP.(Table 5).

Table-6: Comparison of mean periodontal parameters of Group-II at different time periods

Time	Group II				
	Plaque Index (MEAN±SD)	**Gingival index (MEAN±SD)**	**Bleeding on probing (%) (MEAN±SD)**	**Probing pocket depth (mm) (MEAN±SD)**	**Clinical attachment level (mm)(MEAN±SD)**
T0	1.74±2.65	1.58±0.29	34.37±1.27	2.98±0.43	3.35±7.66
T1	1.37±0.22*	1.37±0.12	27.06±1.01*	2.82±0.43	3.16±7.23
T2	1.41±0.28*	1.32±0.13	23.30±9.11*,#	2.78±0.47	3.07±0.75
p value	**0.05**	**2.67**	**0.04**	**1.89**	**1.23**

(*p<0.05 significant compared with T0 with others, #p<0.05 significant compared with T1)

When comparing the mean periodontal parameters of group II at different time periods, it was shown that plaque index decreased significantly at T1 when compared to T0, but again at T2 it was shown that PI score increased when compared to T1 but was not statistically significant. With regard to other parameters, it was shown that GI, PPD and CAL improved from baseline to T2, but significant difference was seen only in relation to BOP at both time periods.

Table-7: Comparison of mean periodontal parameters of Group-III at different time periods

Time	Group III				
	Plaque Index (MEAN±SD)	**Gingival index (MEAN±SD)**	**Bleeding on probing(%) (MEAN±SD)**	**Probing pocket depth(mm) (MEAN±SD)**	**Clinical attachment level(mm) (MEAN±SD)**
T0	1.79±0.34	1.71±0.35	45.09±1.43	3.10±0.52	3.37±0.62
T1	1.47±0.22*	1.44±0.20	33.63±1.43*	3.00±0.45	3.26±0.57
T2	1.35±0.19*	1.31±0.14*,#	23.20±5.96*,#	2.87±0.39*,#	3.10±0.50*,#
p value	**0.05**	**0.04**	**0.04**	**0.05**	**0.05**

(*p<0.05 significant compared T0 with others, #p<0.05 significant compared T1 with others)

Table 7 shows the comparison of mean periodontal parameters of group III at different time periods. It was shown that PI and BOP decreased significantly at T1 and T2 when compared with T0 and improvement in GI, PPD and CAL was significant at T2 only when compared to T0. When compared with T1 statistically significant improvement was seen in relation to BOP, GI, PPD and CAL at T2.

Table-8: Comparison of mean CRP, Iron, TIBC and TSAT values between the groups at T0 time

Groups	CRP(mg/L) (MEAN±SD)	Iron(µg/dl) (MEAN±SD)	TIBC (µg/dl) (MEAN±SD)	TSAT(%) (MEAN±SD)
Group-I	8.06±6.04	38.49±8.94	182.91±38.01	22.02±7.85
Group-II	8.37±6.93	54.31±1.55*	185.05±48.66	29.99±7.77*
Group-III	2.50±0.60*,#	68.08±1.23*,#	241.22±63.08*,#	29.76±8.34*
p value	**0.02**	**0.001**	**0.02**	**0.04**

(*p<0.05 significant compared with Group-I, #p<0.05 significant compared with Group-II)

Comparison of the mean CRP, serum iron, TIBC and TSAT values between the groups at T0 showed that serum iron and TSAT improved significantly in group II and group III whereas CRP and TIBC improved significantly in group III only when compared to group I. CRP decreased, serum iron and TIBC increased significantly in group III when compared to group I.(Table 8).

Table-9: Comparison of mean CRP, Iron, TIBC and TSAT values between the groups at T2 time

Groups	CRP (mg/L) (MEAN±SD)	Iron (µg/dl) (MEAN±SD)	TIBC (µg/dl) (MEAN±SD)	TSAT (%) (MEAN±SD)
Group-I	7.08±5.36	46.77±11.90	204.00±39.99	24.04±8.47
Group-II	7.63±6.12	54.61±16.20*	182.47±47.30*	30.60±7.27*
Group-III	1.09±0.65*,#	70.50±13.98*,#	233.41±63.60*,#	31.89±8.68*,#
p value	**0.02**	**0.002**	**0.04**	**0.03**

(*p<0.05 significant compared with Group-I, #p<0.05 significant compared with Group-II)

Comparison of mean CRP, serum iron, TIBC and TSAT between the groups at T2 showed that the serum iron and TSAT increased statistically significantly in group II and group III when compared to group I. TIBC decreased statistically significantly in group II at T2, but increased statistically significantly at T2 in group III. It was also shown that CRP, Iron, TIBC and TSAT improved significantly in group III when compared to group II(Table 9).

Table-10: Comparison of mean CRP, Iron, TIBC and TSAT values of Group-I between T0 and T2

Time	Group-I			
	CRP (mg/L) (MEAN±SD)	**Iron (µg/dl) (MEAN±SD)**	**TIBC (µg/dl) (MEAN±SD)**	**TSAT (%) (MEAN±SD)**
T0	8.06±6.04	38.49±8.94	182.91±38.01	22.02±7.85
T2	7.08±5.36*	46.77±11.90*	204.00±39.99*	24.04±8.47*
p value	**0.04**	**0.04**	**0.001**	**0.05**

(*p<0.05 significant compared with T0)

Comparison of mean CRP, iron, TIBC and TSAT values of Group I at T0 and T2 showed that CRP decreased, serum iron, TIBC and TSAT increased at T2 from that of T0 and the difference was statistically significant.(Table 10).

Table-11: Comparison of mean CRP, Iron, TIBC and TSAT values of Group-II between T0 and T2

Time	Group-I			
	CRP(mg/L) (MEAN±SD)	**Iron(µg/dl) (MEAN±SD)**	**TIBC (µg/dl) (MEAN±SD)**	**TSAT (%) (MEAN±SD)**
T0	8.37±6.93	54.31±1.55	185.05±48.66	29.99±7.77
T2	7.63±6.12	54.61±16.20	182.47±47.30*	30.60±7.27*
p value	**1.56**	**1.78**	**0.05**	**0.05**

(*p<0.05 significant compared with T0)

Comparison of mean CRP, serum iron, TIBC and TSAT of group II at T0 and T2 showed that serum CRP decreased, serum iron increased, TIBC decreased and TSAT increased at T2 from that of T0, but the difference was statistically significant with respect to TIBC and TSAT alone.(Table 11)

Table-12: Comparison of mean CRP, Iron, TIBC and TSAT values of Group-III between T0 and T2

Time	Group-III			
	CRP (mg/L) (MEAN±SD)	**Iron (µg/dl) (MEAN±SD)**	**TIBC (µg/dl) (MEAN±SD)**	**TSAT (%) (MEAN±SD)**
T0	2.50±0.60	68.08±1.23	241.22±63.08	29.76±8.34
T2	1.09±0.65*	70.50±13.98*	233.41±63.60*	31.89±8.68*
p value	**0.03**	**0.05**	**0.04**	**0.05**

(*p<0.05 significant compared with T0)

When mean CRP, serum iron, TIBC and TSAT of group III was compared at T0 and T2, it was shown that all these parameters improved significantly at T2.(Table 12).

Table-13: Comparison of mean renal and haematological parameters between the groups at T0 time

Groups	Ferritin (ng/mL) (MEAN±SD)	Hb (gm/dL) (MEAN±SD)	ESR (mm/Hr) (MEAN±SD)	Albumin (gm/dL) (MEAN±SD)
Group-I	458.78±2.55	9.04±1.58	66.40±19.49	3.92±0.31
Group-II	427.80±3.28*	8.71±1.68*	56.05±19.73*	4.11±0.36*
Group-III	51.41±2.11*,#	11.71±1.35*,#	19.70±8.26*,#	4.06±0.19
p value	**0.001**	**0.04**	**0.001**	**0.05**

(*p<0.05 significant compared with Group-I, #p<0.05 significant compared with Group-II)

Comparison of serum ferritin, Hb, ESR and serum albumin between the groups at T0 showed that statistically significant difference was seen in relation to serum ferritin, Hb and ESR in group II and Group III compared with group I and also statistically significant difference was seen in serum albumin with respect to group II only when compared to group I. at baseline, it was also shown that there was statistically significant difference in serum ferritin, Hb and ESR in group III when compared to group II.(Table 13)

Table-14: Comparison of mean renal and haematological parameters between the groups at T2 time

Groups	Ferritin (ng/mL) (MEAN±SD)	Hb (gm/dL) (MEAN±SD)	ESR(mm/Hr) (MEAN±SD)	Albumin (gm/dL) (MEAN±SD)
Group-I	500.34±2.96	9.54±1.95	59.45±19.56	3.98±0.21
Group-II	469.81±3.22*	8.76±1.65*	53.80±23.41*	4.04±0.25
Group-III	47.36±2.23*,#	12.04±1.04*,#	17.45±6.41*,#	4.06±0.22
p value	**0.001**	**0.04**	**0.002**	**1.67**

(*p<0.05 significant compared with Group-I, #p<0.05 significant compared with Group-II)

Table 14 shows that statistically significant difference in serum ferritin, Hb and ESR was present at T2 in group II and Group III when compared to Group I and also statistically significant difference was seen in group III at T2 when compared to that of Group II.(Table 14)

Table-15: Comparison of mean renal and haematological parameters within the Group-I at different time periods

Groups	Group-I			
	Ferritin (ng/mL) (MEAN±SD)	**Hb(gm/dL) (MEAN±SD)**	**ESR (mm/Hr) (MEAN±SD)**	**Albumin (gm/dL) (MEAN±SD)**
T0	458.78±2.55	9.04±1.58	66.40±19.49	3.92±0.31
T2	500.34±2.96*	9.54±1.95	59.45±19.56*	3.98±0.21
p value	**0.001**	**1.89**	**0.03**	**2.45**

(*p<0.05 significant compared with T0)

Within Group I at T0 and T2, it was shown that serum ferritin increased and ESR decreased significantly at T2 when compared to baseline. It was also noted that Hb and serum albumin increased at T2 from that of baseline, eventhough it was not statistically significant.(Table 15).

Table-16: Comparison of mean renal and haematological parameters within the Group-II at different time periods

Groups	Group-II			
	Ferritin (ng/mL) (MEAN±SD)	**Hb (gm/dL) (MEAN±SD)**	**ESR(mm/Hr) (MEAN±SD)**	**Albumin (gm/dL) (MEAN±SD)**
T0	427.80±3.28	8.71±1.68	56.05±19.73	4.11±0.36
T2	469.81±3.22*	8.76±1.65	53.80±23.41*	4.04±0.25
p value	**0.003**	**1.45**	**0.05**	**1.19**

(*p<0.05 significant compared with T0)

Comparison of serum ferritin, Hb, ESR and serum albumin within group II at different time periods showed that serum ferritin increased and ESR decreased at T2 from that of baseline and the difference was statistically significant (Table 16).

Table-17: Comparison of mean renal and haematological parameters within the Group-III at different time periods

Groups	Group-III			
	Ferritin (ng/mL) (MEAN±SD)	**Hb (gm/dL) (MEAN±SD)**	**ESR (mm/Hr) (MEAN±SD)**	**Albumin (gm/dL) (MEAN±SD)**
T0	51.41±2.11	11.71±1.35	19.70±8.26	4.06±0.19
T2	47.36±2.23*	12.04±1.04	17.45±6.41*	4.06±0.22
p value	**0.04**	**2.89**	**0.05**	**1.56**

(*p<0.05 significant compared with T0)

Table 17 shows the comparison of serum ferritin, Hb, ESR and serum albumin in group III at baseline and T2, it was shown that serum ferritin and ESR decreased at T2 from that of T0 and the difference was statistically significant.

Table-18: Comparison of mean creatinine and GFR values between the groups at T0 and T2 time

Groups	Creatinine at T0 time (MEAN±SD)	Creatinine at T2 time (MEAN±SD)	GFR at T0 time (MEAN±SD)	GFR at T2 time (MEAN±SD)
Group I	8.71±1.98	8.46±2.31	7.40±1.98	7.70±2.69
Group II	6.69±2.68*	8.17±3.95	11.20±6.32*	9.45±5.94*
Group III	0.79±0.11*,#	0.74±0.10*,#	99.55±1.47*,#	105.40±1.46*,#
p value	**0.001**	**0.001**	**0.001**	**0.001**

(*p<0.05 significant compared with Group-I, #p<0.05 significant compared with Group-II)

Table 18 shows mean serum creatinine and GFR between the groups at T0 and T2. It showed that statistically significant difference was seen in serum creatinine and GFR in group II and Group III when compared to Group I at T0 and also statistically significant difference was seen in group III compared to group II in terms of serum creatinine and GFR at T0. Table 18 also shows that statistically significant difference in GFR was present in group II when compared to Group I and statistically significant difference was seen in group III with respect to serum creatinine and GFR compared to Group I and Group II at T2.

Table-19: Comparison of mean creatinine and GFR values within the groups at different time periods

Time	Group-I		Group-II		Group-III	
	Creatinine (MEAN±SD)	**GFR (mL/ min/1.73m^2) (MEAN±SD)**	**Creatinine (MEAN±SD)**	**GFR(mL/ min/1.73m^2) (MEAN±SD)**	**Creatinine (MEAN±SD)**	**GFR (mL/ min/1.73m^2) (MEAN±SD)**
T0	8.71±1.98	7.40±1.98	6.69±2.68	11.20±6.32	0.79±0.11	99.55±1.47
T2	8.46±2.31	7.70±2.69	8.17±3.95	9.45±5.94	0.74±0.10	105.40±1.46*
p value	**1.67**	**1.45**	**0.06**	**0.06**	**2.45**	**0.05**

(*p<0.05 significant compared with T0)

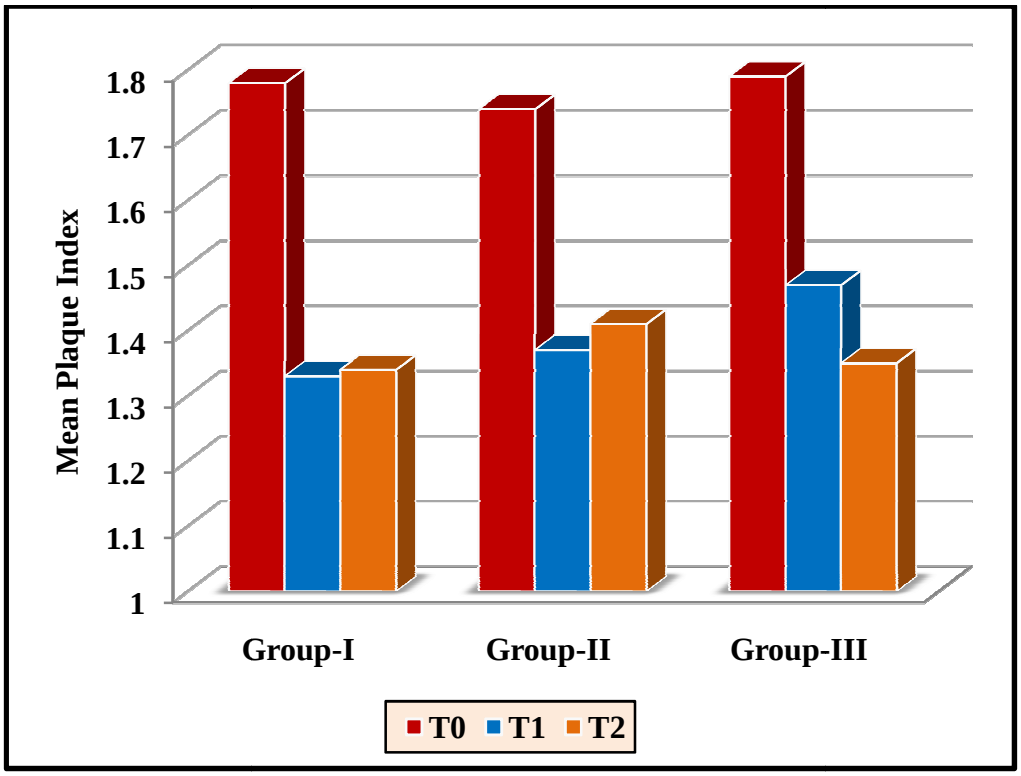
Table 19 shows the comparison of serum creatinine and GFR within the group at T0 and T2. In Group I it was noted that serum creatinine decreased and GFR increased at T2 from that of baseline, but it was not statistically significant.

In Group II Table 19 shows that serum creatinine and GFR decreased at T2 from that of baseline, but that was also not statistically significant.

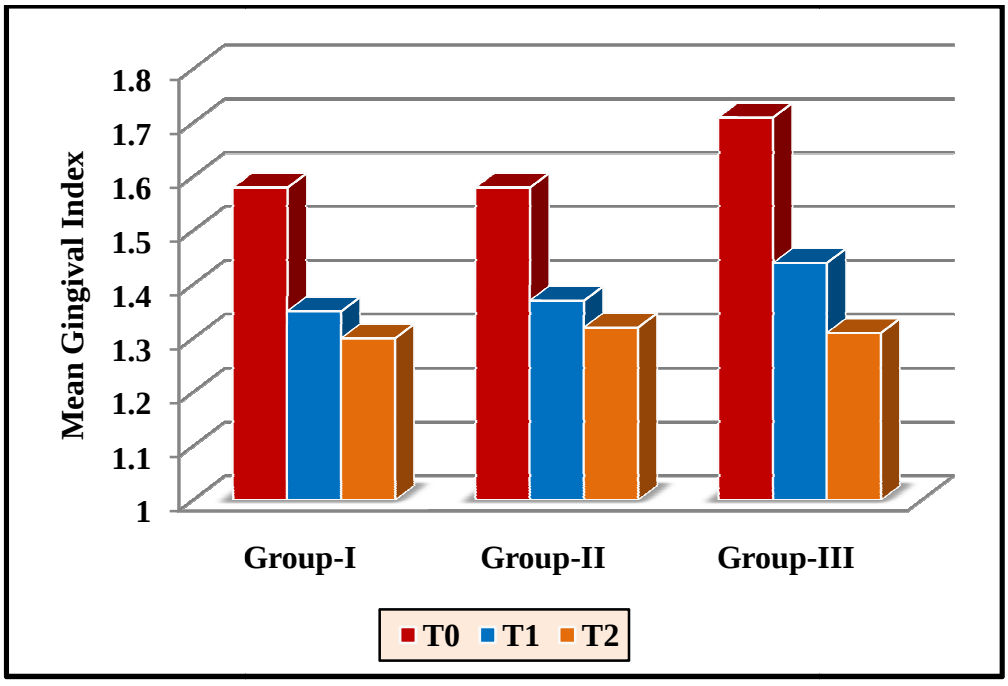
In Group III it was shown that serum creatinine decreased and GFR increased at T2 from that of baseline, but the increase in GFR alone was statistically significant.

GRAPHS

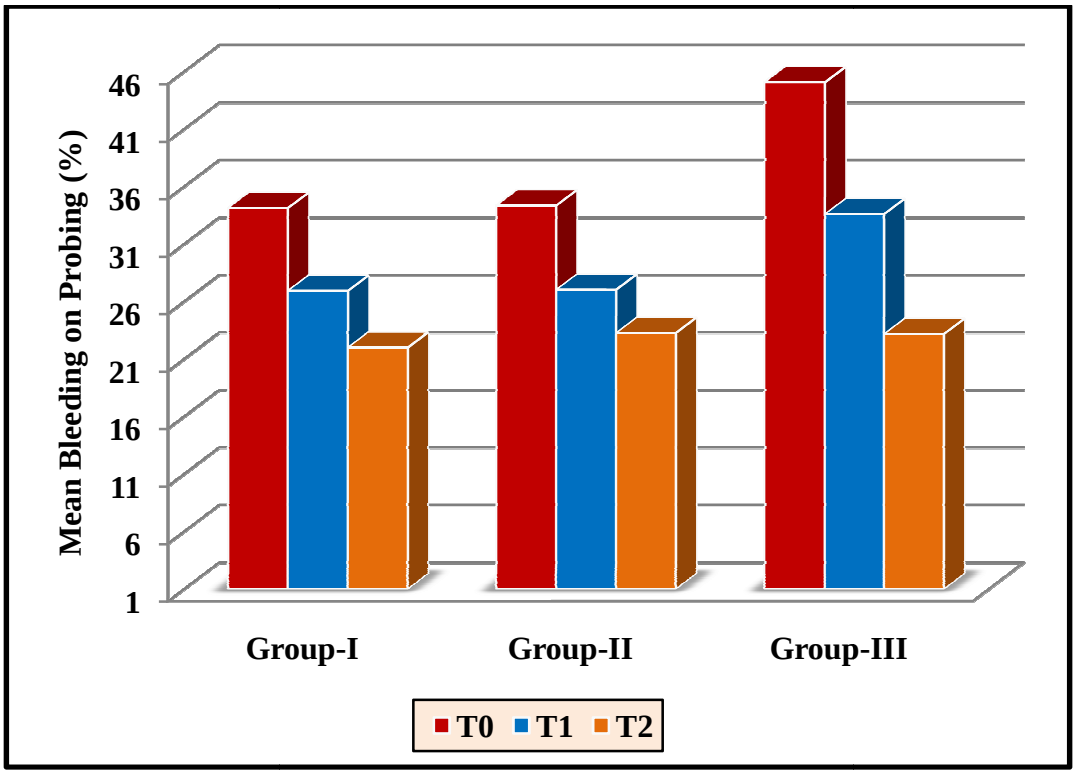
Graph-1. Comparison of mean Plaque Index values of different groups



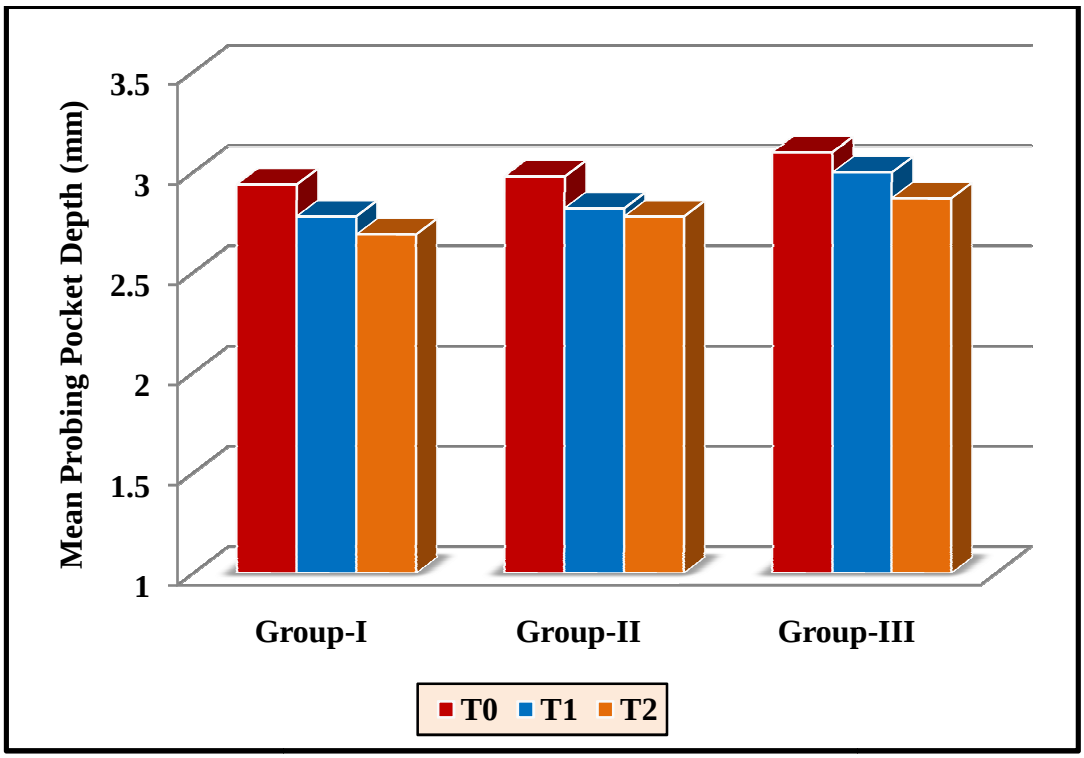
Graph-2: Comparison of mean Gingival Index between the groups



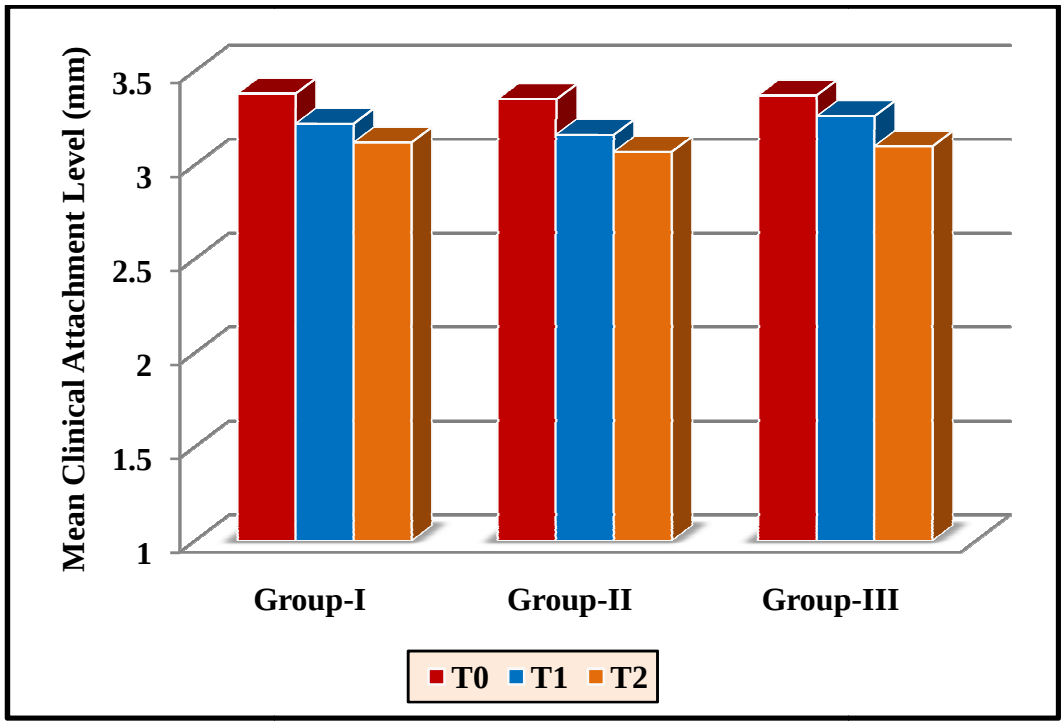
Graph-3: Comparison of mean Bleeding on Probing values of different groups



Graph-4: Comparison of mean Probing Pocket Depth between the groups



Graph-5: Comparison of mean Clinical Attachment Level between the groups



DISCUSSION

Chronic kidney disease occurs due to a slow and progressive decline of kidney function. It happens gradually over a period of weeks, months or years as the kidneys slowly stop working, leading to End stage renal disease (ESRD).[77] Both chronic kidney disease as well as renal replacement therapy that includes haemodialysis, peritoneal dialysis, or renal transplantation can affect periodontal tissue.[74] Periodontal disease is an inflammatory disease that affects the supporting tissues of the teeth mainly due to the interaction between specific bacterial species and the host immune response in disease susceptible individuals.[78] Local destruction that occurs in periodontitis can in turn lead to the production of inflammatory mediators such as C-reactive protein (CRP), Interleukin-6, Interleukin-1, TNF-α and Prostaglandin-$E_{2.}$ Periodontitis has been reported to be associated with increased systemic inflammatory burden even in end stage renal disease patients on haemodialysis maintenance therapy which in turn may exacerbate the existing metabolic disorder which include hyperglycemia, hypertension and dyslipidemia in these patients.[69, 79, 80] These low grade systemic inflammation, such as due to periodontitis has been reported to increase the risk for cardiovascular events.[59] However, periodontal diseases are treatable and can be considered as a modifiable risk factor.[78] Treating periodontal disease may decrease the proinflammatory state in this population. It may also improve the oral discomfort and improve the nutritional status.[81] In a study it was reported that following periodontal therapy the mean CRP level and erythrocyte sedimentation rate decreased and haemoglobin level increased in haemodialysis patients, suggesting a decrease in inflammatory state.[82] Therefore, non surgical periodontal therapy was found to eliminate periodontal infection as well as improve systemic status, both of which are involved in the progression of ESRD.[70, 83]

Plaque index (PI) provides a quick and simple method of monitoring a patient's ability to control plaque from the tooth surface. Our study results showed that the Plaque Index score was reduced in the initial one month postoperative(T1) after which at 3 months postoperative(T2) there was a slight increase in PI in the haemodialysis groups (Group I and Group II)(Graph 1). This shows the poor compliance to the oral hygiene instructions given to the haemodialysis group. This is in accordance with the study conducted by Wehmeyer et al, 2013[73] in which it was shown that there was decrease in the PI at 3 months post operative after which at 6 months there was an increase in haemodialysis group, which showed poor maintenance in haemodialysis patients. Fang et al, 2015[69] also showed similar results in which after initial periodontal therapy the periodontal parameters were found to be reduced and then during the follow up it was found to be increased in the haemodialysis group which is in support to the results of our study. But the decrease in PI score was statistically significant in both Group I and Group II from baseline to 3 months postoperative.

GI is a method of recording the clinical severity of gingival inflammation. GI was also found to be reduced from baseline to 3 months post-treatment, but was not statistically significant in Group I and II (Graph 2). BOP is a simple and reliable indicator of gingival inflammation, which was found to be reduced in Group I and Group III significantly from baseline to 3 months post-treatment (Graph 3). Probing pocket depth (PPD) and Clinical attachment level (CAL) was also found to be reduced, though not statistically significant from baseline to T2 in Group 1 and Group II (Graph 4 and Graph 5).

In the systemically healthy chronic periodontitis group, all the clinical parameters (PI, GI, BOP, PPD and CAL) decreased significantly at 3 months postoperatively (Table 7) which is similar to the results of the study conducted by Radafshar et al. 2010,[81] Graziani et al. 2010,[83] Artese et al. 2010,[76] Fang et al. 2015[69]. All these studies showed a significant reduction in the clinical parameters postoperatively in systemically healthy chronic periodontitis patients. This shows good compliance to the oral hygiene instruction given in Group III when compared to that of Group I and Group II.

C- reactive protein level in our study results showed that postoperatively there was statistically significant reduction in Group I and Group III(8.06±6.04 (mg/L) to 7.08±5.36 mg/L,p= 0.04 and 2.50±0.60 mg/L to 2.09±0.65 mg/L, p=0.03, respectively) (Table 8 and Table 9) whereas the reduction in the CRP level in Group II was not statistically significant, this shows that the inflammation in Group II (Chronic kidney disease patients undergoing haemodialysis for more than a year) was more severe than that of Group I (Chronic kidney disease patients undergoing haemodialysis for less than a year). Also at baseline, it was shown that CRP level was more in Group II than that of Group I (8.06±6.04 mg/L and 8.37±6.93 mg/L, respectively) (Table 8), which might be due to the severity in inflammation in group II than in Group I. This is in accordance with that study by Yazdi et al, 2013 which showed reduction in CRP level in maintenance haemodialysis patients after 2 months of periodontal therapy (9.369±11.25mg/L to 5.628±4.79 mg/L).[74] Study by Fang et al, 2015 also showed that compared to the periodontitis patients who did not receive any treatment, CRP values decreased in periodontitis patients who were under maintenance haemodialysis. One of the best predictor of cardiac and all-cause

mortality in ESRD patients on haemodialysis are CRP values.[84] Inflammation arising from infections associated with destructive periodontal diseases appears to contribute to elevated values of systemic CRP. Since destructive periodontal disease is a treatable condition, its management can bring about a decrease in CRP values and the associated risk of atherosclerotic complications in ESRD patients undergoing haemodialysis.[78]

Transferrin saturation is calculated as a percentage of serum iron and total iron binding capacity (TIBC), which corresponds to the circulating iron. The TIBC reflects transferrin, which is the protein to which all iron in the blood is bound. Due to inflammatory stimulation, the iron that is in the reticuloendothelial storage gets locked up and is not released to transferrin. As a result of which, TSAT is low, despite a normal or elevated ferritin.[85] There will be decrease in the transferrin saturation which leads to decrease in erythropoiesis and contributes to the development of anemia of chronic diseases.[86] In our study it was shown that 3 months after periodontal treatment, transferrin saturation was found to increase in all three group significantly ($p<0.05$) (Table 8 and Table 9). This was similar to the results in the study by Vilele EM et al.[32]

Serum ferritin is an acute phase reactant and has been reported to be increased in inflammation, autoimmune disease, chronic infection and liver diseases.[70] In our study it was shown that serum ferritin was found to be significantly more in haemodialysis group when compared to the serum ferritin level in chronic periodontitis at baseline. In group I and group II, serum ferritin level was found to be increased at 3 months postoperatively (458.78±2.55 ng/mL to 500.34±2.96 ng/mL and 427.80±3.28 ng/mL to 469.81±3.22 ng/mL, respectively) (Table 15 and

Table 16), whereas in systemically healthy chronic periodontitis group, serum ferritin reduced 3 months postoperatively. Study conducted by Chakraborty et al, 2014[70] showed that after 3 months of nonsurgical periodontal therapy, serum ferritin was found to be reduced in systemically healthy chronic periodontitis patients which was similar to that of our study. During acute phase response, proinflammatory cytokines interleukin 1 beta and tumour necrosis factor alpha (TNF-α) increase the synthesis of various subunits of ferritin. Inflammation induced hyperferritinemia can block the iron mobility and hence be harmful under chronic inflammation by leading to refractory anemia, such as in chronic kidney disease or other chronic disease states.[87] This can be the reason for the increase in serum ferritin in haemodialysis group inspite of improvement in the clinical parameters.

Lower haemoglobin levels were seen at baseline in all the three groups, which might be due to due to proinflammatory cytokines, which act as mediators in suppressing erythropoiesis from bone marrow.[70] The present study also showed that at 3 months post-treatment, there was an increase in the Haemoglobin level which was not statistically significant ($p>0.05$). It also showed a statistically significant reduction in the ESR rate in all the three groups 3 months post-operatively ($p<0.05$) (Table 13 and Table 14). ESR is considered as a valuable parameter for inflammatory process. The results regarding haemoglobin and ESR was also similar to that of study conducted by Musalaiah et al,[31] Agarwal N et al.[88] Improvement in ESR postoperatively was due to a reduction of the periodontal inflammation and the inflammatory markers.[89] Non surgical periodontal treatment can improve the anemic status of patients on chronic periodontitis and thereby bring about improvement in the haematological parameters in turn.

Albumin is a negative acute phase reactant, which tends to be lower in case of inflammation.[90] In our study it was shown that the serum albumin level was found to increase 3 months postoperatively in Group I (3.92±0.31 gm/dL to 3.98±0.21 gm/dL, not statistically significant, p=2.45) and was found to decrease in the Group II after 3 months postoperatively(4.11±0.36 gm/dL to 4.04±0.25 gm/dL, not statistically significant, p=1.19) whereas it was almost at the same level in the Group III patients from baseline to T2 (4.06±0.19 gm/dL to 4.06±0.22 gm/dL) (Table 13 and Table 14). In our study there was no much statistically significant difference in the serum albumin level in the groups between baseline and 3 months postoperatively. Our results are similar to the study by Wehmeyer et al.[73] In their study they reported that the intensive periodontal treatment was not associated with an improvement in serum albumin level at either 3 or 6 months of follow up. Two important factors that regulate albumin synthesis are nutritional intake and illness.[91] Albumin levels have been reported to decrease inflammatory disorders and other illness. Factors regulating serum albumin are similar in patients with and without chronic kidney disease. This might be the reason, that serum albumin levels did not vary much in chronic periodontitis group and chronic kidney disease patients undergoing haemodialysis.

Serum creatinine level decreased in Group I and group III, but in group II there was an increase in the serum creatinine in Group II from baseline to 3 months post periodontal treatment, but the difference was not statistically significant. With regard to GFR also, in Group I and Group III increased (7.40±1.98 mL/min/1.73m^2 to 7.70±2.69 mL/min/1.73m^2, p=1.45 and 99.55±1.47mL/min/1.73m^2 to 105.40±1.46 mL/min/1.73m^2 *, p=0.05* respectively), whereas decreased in Group

II (11.20±6.32 mL/min/1.73m^2 to 9.45±5.94 mL/min/1.73m^2, p=0.06) (Table 19). Artese et al, 2010 in his study reported that serum creatinine decreased and GFR rate increased in predialytic patients and also in systemically healthy chronic periodontitis patients.[76] Graziani et al, 2010 in a study have shown an improvement in the GFR in chronic periodontitis patients after nonsurgical periodontal therapy.[83] In Chronic kidney disease, there is steady and continuous decrease in renal clearance or GFR, which leads to gathering of creatinine, urea and other substances in the blood.[77] Inflammation in periodontal disease can cause increase in inflammatory markers, which can lead to progression of kidney disease and a further increase in inflammatory markers. Therefore a reduction of inflammation and the consequent improvement of endothelial function associated with periodontal therapy can in turn have an impact on the kidney microcirculation with a subsequent more effective filtration.[83]

Demmer RT et al.,[92] de Freitas et al.,[93] Ioannidou E et al.,[94] in a systematic review reported that there was reduction in systemic CRP following periodontal treatment in periodontitis patients. Chambrone L et al. in a systematic review also gave quite a consistent evidence to support the positive association between periodontitis and chronic kidney disease, as well as the positive effect on periodontal therapy on estimated GFR.[95]

The present study has limitations that need to be clearly discussed which include use of antibiotics in chronic kidney disease patients undergoing haemodialysis before non-surgical periodontal therapy which could have affected the clinical, renal and haematological parameters. Another limitation includes relatively small sample size and short duration of follow up (3 months) of the study.

SUMMARY & CONCLUSION

The objective of the present study was to evaluate the effect of nonsurgical periodontal therapy on clinical parameters (at baseline, 1 month and 3 months), renal [C-reactive protein (CRP), Transferrin Saturation (TSAT), Serum ferritin] haematological parameters (haemoglobin, Erythrocyte Sedimentation Rate) (at baseline and 3 months) in systemically healthy periodontitis patients and chronic kidney disease patients undergoing haemodialysis of varying duration. The clinical parameters (Plaque index, Gingival index, Bleeding on Probing, Probing Pocket Depth, Clinical Attachment Level) was found to be reduced in both the groups after periodontal therapy. It was also noted that CRP levels decreased and TSAT increased in all the three groups, serum ferritin was found to increase in chronic kidney disease patients undergoing haemodialysis, where as decreased in systemically healthy chronic periodontitis patients. Thus we conclude that nonsurgical periodontal therapy can contribute to improvement in acute phase reactants like CRP and iron indices in systemically healthy chronic periodontitis patients and in chronic kidney disease patients undergoing haemodialysis. Further studies with larger sample size and longer duration of follow up are required to validate the observations of the present study.

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ANNEXURE

Annexure 1

SREE MOOKAMBIKA INSTITUTE OF DENTAL SCIENCES

KULASEKHARAM, KANYAKUMARI DIST., TAMIL NADU, INDIA.

INSTITUTIONAL RESEARCH COMMITTEE

Certificate

This is to certify that the research project protocol, ***Ref no. 18/06/2015*** titled, ***"Effect of non surgical periodontal therapy on C-reactive protein and iron indices in haemodialysis patients with chronic periodontitis"*** submitted by ***Dr. Sheethel Menon V., II Year MDS, Department of Periodontics and Oral Implantology*** has been approved by the Institutional Research Committee at its meeting held on 15th ***June 2015.***

Convener
Dr. T. Sreelal

Secretary
Dr. Pradeesh Sathyan

Annexure 2

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES

(Kulasekharam (K.K District, TN)-629161, Phone No: 04651-280866, Fax No: 280740)

Institutional Human Ethics Committee (IHEC)

{CDSCO Reg No: ECR/446/Inst/TN/2013}

Ref. No: SMIMS/IHEC/2015/A/05 **Date: 17th February 2016**

CERTIFICATE

This is to certify that the Research Protocol Ref. No. **SMIMS/IHEC/2015/A/05** entitled "Effect of Nonsurgical Periodontal Therapy on C-Reactive Protein and Iron Indices in Haemodialysis Patients with Chronic Periodontitis" submitted by Dr. Sheethel Menon. V, Postgraduate of Department of Periodontics and Oral Implantology, SMIDS has been approved by the Institutional Human Ethics Committee at its meeting held on 10th December 2015.

IHEC Date 17/2/16

17/2/16

Dr. Rema Menon. N
Member Secretary
Institutional Human Ethics Committee
Professor and HOD of Pharmacology
SMIMS, Kulasekharam (K.K District)
Tamil Nadu-629161

[This Institutional Human Ethics Committee is organized and is operating according to the requirements of ICH-GCP/GLP guidelines and requirements of the Amended Schedule-Y of Drugs and Cosmetics Act, 1940 and Rules 1945 of Government of India.]

Annexure 3

PATIENT INFORMATION SHEET

Dear Volunteers,

We welcome you and thank you for your keen interest in participation in this research project. Before you participate in this study, it is important for you to understand why this research is being carried out. This form will provide you all the relevant details of this research. It will explain the nature, the purpose, the benefits, the risks, the discomforts, the precautions and the information about how this project will be carried out. It is important that you read and understand the contents of the form carefully. This form may contain certain scientific terms and hence, if you have any doubts or if you want more information, you are free to ask the study personnel or the contact person mentioned below before you give your consent and also at any time during the entire course of the project.

1. Name of the Principal Investigator: Dr. Sheethel.Menon.V

Post graduate student,
Department of Periodontics
SMIDS,Kulasekaram 629161
Cell: 9445678206
sheethel.menon@gmail.com

2. Name of the Guide: Dr Arun Sadasivan, MDS

Professor,
Department of Periodontics
SMIDS, Kulasekharam 629161
Cell: 9847246961
sadasivan_arun@hotmail.com

3. Name of the Co-Guide: Dr. Elizabeth Koshi, MDS

Head of the Department,
Department of Periodontics,
SMIDS, Kulasekharam 629161
Cell: 09447154335
elizabethkoshi_dr@yahoo.com

4. Institute : Sree Mookambika Institute of Dental Sciences, Kulasekharam

5. Title of the study: 'Effect of nonsurgical periodontal therapy on C-reactive protein and iron indices in haemodialysis patients with chronic periodontitis' .

6. Background information:

From the review of several scientific publications from 2009 to 2014

7. Aims and Objectives:

The aim of the study is to evaluate the effect of scaling and root planing on changes in the C-reactive protein and iron indices in chronic periodontitis patients and in renal patients undergoing haemodialysis with chronic periodontitis

8. Scientific justification of the study:

Non-Surgical Periodontal therapy can bring about decrease in the bacterial load and improve the periodontal status which can bring about decrease in the CRP level and also increase the level of serum iron, serum ferritin and TIBC in haemodialysis patients with chronic periodontitis and decreases periodontal destruction. Periodontal Therapy is able to reduce acute-phase proteins levels and systemic inflammatory markers, which are involved in the progression of renal disease.[13]Thus periodontal therapy should also be able to exert beneficial effects on the function of the kidney.

9. Procedure for the study:

- Once you are enrolled into the study a roll number will be implemented to represent the name.
- When you visit the clinic for periodontal examination, under sterile condition 5ml blood samples will be collected using a syringe and 23 gauge needle and sent to the laboratory for estimation of CRP level and the level of serum iron, serum ferritin and TIBC.
- Then you will be undergoing periodontal examination that involves the evaluation of gums as well as the amount of plaque present on the teeth.
- Then you will be undergoing scaling and root planing(Cleaning of teeth).
- You will be recalled after 1 month and periodontal status will be re-evaluated and again 5 mL of blood samples will be collected for the above mentioned analysis.

10. Expected risks for the participants:

Pain, ecchymosis are expected very rarely.

11. Expected benefits of research for the participants:

There is no direct benefit to you. This study will enhance our knowledge aboutthe effect of nonsurgical periodontal therapy onthe level of CRP level and serum iron, serum ferritin and TIBC in chronic periodontitis patients and in renal patients undergoing haemodialysis with chronic periodontitis

12. Maintenance of confidentiality:

- You have the right to confidentiality regarding the privacy of your medical information(Personal details, results of physical examinations, investigations, and your medical history).
- By signing this document, you will be allowing the research team investigators, other study Personnel, sponsors, institutional ethics committee and any person or agency required by law to view your data, if required.
- The results of clinical tests and therapy performed as part of this research may be included in your medical record.
- The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your identity.

13. Why have I been chosen to be in this study?

You were selected from a pool of patients in outpatient Department of Periodontics, Sree Mookambika Institute of Dental Sciences, Kulasekharam and Department of Nephrology, Sree Mookambika Institute of Medical Sciences, Kulasekharam. You will be informed of this study and will be invited to participate. You have been invited to this study because you fulfill our inclusion criteria by being a systemically healthy subject between the age of 30-65yrs. No invasive procedure is done that harm your health and it helps in diagnosis and will be helpful for the society.

14.How many people will be in the study? Sixty individuals

15. Agreement of compensation to the participants (In case of a study related injury): Patient will be taken care in case of complication and medical treatment will be provided in the institution at the expense of the principle investigator.

16. Anticipated prorated payment, if any, to the participant(s) of the study:

Nil

17. Can I withdraw from the study at any time during the study period?

The participation in this research is purely voluntary and you have the right to withdraw from this study at any time during the course of the study without giving any reasons.

18. If there is any new findings/information, would I be informed?

Yes

19. Expected duration of the participant's participation in the study:

Six Months

20. Any other pertinent information: No other information

21. Whom do I contact for further information? For further information, free feel to contact:

Dr. Sheethel Menon.V
Post graduate student,
Department of Periodontics,
SMIDS,
Kulasekaram 629161
Ph: 9445678206
sheethel.menon@gmail.com

Place:

Date:

Signature of the participant **Signature of Principal Investigator**

Annexure 3

സമ്മത പത്രം - ഭാഗം - 1
പഠനവുമായി സഹകരിക്കുന്ന വ്യക്തികളുടെ അറിവിലേയ്ക്ക്

പ്രിയപ്പെട്ട സന്നദ്ധ സേവകൻ / സേവക,

ഞങ്ങൾ നിങ്ങളെ സ്വാഗതം ചെയ്യുന്നു. അതോടൊപ്പം ഈ പഠനവുമായി സഹകരിക്കാനുള്ള സന്നദ്ധതയോട് നന്ദി രേഖപ്പെടുത്തുന്നു. നിങ്ങൾ ഈ പഠനത്തിൽ പങ്കെടുക്കുന്നതിനു മുൻപ് ഈ പഠനം എന്തിനാണ് നടത്തപ്പെടുന്നത് എന്ന് അിറയേണ്ടതുണ്ട്. അതിനാൽ ഈ ഫോറത്തിൽ ഗവേഷണ പഠനത്തിന്റെ വിവരങ്ങളും മറ്റും വിശദമായി രേഖപ്പെടുത്തിയിരിക്കുന്നു. ഈ പഠനത്തിന്റെ രീതി, ഉദ്ദേശം, പ്രയോജനം, അപകടസാദ്ധ്യത, ക്ലേശം, മുൻകരുതൽ, എങ്ങനെ ഈ പഠനം മുൻപോട്ടു കൊണ്ടുപോകുന്നു എന്നിങ്ങനെ എല്ലാ വിവരങ്ങളും ഫോറത്തിൽ രേഖപ്പെടുത്തിയിരിക്കുന്നു. സദയം ഈ വിവരങ്ങൾ വായിച്ചു മനസ്സിലാക്കുവാൻ അഭ്യർത്ഥിക്കുന്നു. ഈ വിവരങ്ങളിൽ ശാസ്ത്രപരമായ പദങ്ങൾ ഉള്ളതിനാൽ സംശയനിവാരണത്തിനു പ്രധാന പഠനകർത്താവിനോടോ താഴെ രേഖപ്പെടുത്തിയിരിക്കുന്ന വ്യക്തികളോടോ ഫോറം ഒപ്പിടുന്നതിനു മുൻപോ അല്ലെങ്കിൽ ഈ പഠനത്തിന്റെ കാലാവധി തീരുന്നതുവരേയോ സമീപിക്കാവുന്നതാണ്.

1. മുഖ്യ ഗവേഷകൻ :

ഡോ. ശീതൾ മേനോൻ.വി
പോസ്റ്റുഗ്രാജുവേറ്റ്
ഡിഷാർട്ട്മെന്റ് ഓഫ് പെരിയോഡോന്റിക്സ്
ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് ഡെന്റൽ സയൻസ്,
കുലശേഖരം - 629 161.
ഫോൺ: 9445678206

2. പ്രധാന മാർഗ്ഗദർശി :

ഡോ.അരുൺ സദാശിവൻ
പ്രൊഫസർ
ഡിഷാർട്ട്മെന്റ് ഓഫ് പെരിയോഡോന്റിക്സ്
ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് ഡെന്റൽ സയൻസസ്,
കുലശേഖരം.
ഫോൺ: 9847246961

3. **സഹ മാർഗ്ഗദർശി** :

ഡോ. എലിസബെത്ത് കോശി
ഹെഡ് ഓഫ് ദി ഡിഷാർട്ട്മെന്റ്,
ഡിഷാർട്ട്മെന്റ് ഓഫ് പെരിയോഡോന്റിക്സ്
ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് ഡെന്റൽ സയൻസസ്,
കുലശേഖരം.
ഫോൺ: 9447154335

ഡോ. ബീനാ ഉണ്ണികൃഷ്ണൻ
അസോസിയേറ്റ് പ്രൊഫസർ
ജനറൽ മെഡിസിൻ വിഭാഗം,
ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് മെഡിക്കൽ സയൻസസ്.
കുലശേഖരം
Mob: 9947777615
Email: beena.unnikrishnan@gmail.com

4. ഇൻസ്റ്റിറ്റ്യൂട്ട് :

ശ്രീ. മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് ഡെന്റൽ സയൻസസ്
പടനിലം, കുലശേഖരം,
കന്യാകുമാരി - 629 161.
തമിഴ്നാട്.

Annexure 3

5. പഠനത്തിന്റെ ശീർഷകം :

സ്കെയ്ലിംഗ് & റൂട്ട് പ്ലേനിംഗ് ചികിത്സയ്ക്ക് മുൻപും ശേഷവും പെരിഡോന്റൈറ്റിസ് രോഗികളിലും, ഹീമോഡയാലിസിസ് ചെയ്യുന്ന പെരിഡോന്റൈറ്റിസ്രോഗികളിലും ഉണ്ടാകുന്ന സി-റിയാക്ട്ടിവ് പ്രോട്ടീനിലും ഇരുമ്പ് സൂചികകൾ - ലും വരുന്ന വ്യത്യാസം.

6. പശ്ചാത്തല വിവരം ?

2009 മുതൽ **2014** വരെയുള്ള ശാസ്ത്രീയ പ്രസിദ്ധീകരണങ്ങളിൽ നിന്നും ശേഖരിച്ച വിവരണങ്ങൾ.

7. ലക്ഷ്യങ്ങളും ഉദ്ദേശങ്ങളും

സ്കെയ്ലിംഗ് & റൂട്ട് പ്ലേനിംഗ് ചികിത്സയ്ക്ക് മുൻപും ശേഷവും പെരിഡോന്റൈറ്റിസ് രോഗികളിലും, ഹീമോഡയാലിസിസ് ചെയ്യുന്ന പെരിഡോന്റൈറ്റിസ്രോഗികളിലും ഉണ്ടാകുന്ന സി-റിയാക്ട്ടിവ് പ്രോട്ടീനിലും ഇരുമ്പ് സൂചികകൾ -ലും വരുന്ന വ്യത്യാസം.

8. ഗവേഷണം നടത്താനുള്ള ന്യായീകരണം

സ്കെയ്ലിംഗ്& റൂട്ട് പ്ലേനിംഗ് ചികിത്സ ബാക്ടീരിയകളുടെ അളവു കുറയ്ക്കുകയും അതുവഴി സി-റിയാക്ട്ടിവ് പ്രോട്ടീൻ കുറയുകയും സീറത്തിലെഅയർൺ, ട്ടോട്ടൽ അയർൺ ബൈൻഡിംഗ് കപ്പാസ്സിറ്റി, ഫെറിട്ടിൻ അളവ് കൂടുകയും പെരിഡോൻഷ്യത്തിന്റെ ആരോഗ്യത്തിനെ മെച്ചപ്പെടുത്തുകയും ചെയ്യുന്നു

9. പഠന രീതി

- പഠനത്തിന്റെ ഭാഗമായി വ്യക്തിക്ക് ഒരു നമ്പർ നൽകുന്നതാണ്.
- നിങ്ങളുടെ മുഴുവൻ വിവരങ്ങളുമടങ്ങിയ കേസ് ചാർട്ട് രേഖപ്പെടുത്തുന്നതാണ്. ആവശ്യമെങ്കിൽ പരിശോധന നടത്തപ്പെടുന്നതാണ്. ആവശ്യാനുസരണമായി ചിത്രങ്ങൾ എടുക്കപ്പെടുന്നതാണ്.
- പെരിയോഡോൻഷ്യത്തിന്റെ പരിശോധനവേളയിൽ 5 എം.എൽ രക്തം ഒരു സിറിഞ്ചും നീടിലും ഉപയോഗിച്ച് ശേഖരിച്ച് സി-റിയാക്ട്ടിവ് പ്രോട്ടീൻ,സീറത്തിലെഅയർൺ, ട്ടോട്ടൽ അയർൺ ബൈൻഡിംഗ് കപ്പാസ്സിറ്റി, ഫെറിട്ടിൻ അളവ്കൺടുപിടിക്കുന്നതായിരിക്കും.
- രക്തം ശേഖരിക്കുന്നത് രാവിലെ 8 മണിക്കും 10മണിക്കും മദ്ധ്യേയായിരിക്കും.
- അതിനുശേഷം സ്കെയിലിംഗ് & റൂട്ട്പ്ലാനിംഗ് എന്ന ചികിത്സ ചെയ്യുന്നതായിരിക്കും.
- ഒരുമാസത്തിനുശേഷം നിങ്ങളുടെ പെരിയോഡോൻഷ്യത്തെ പരിശോധിക്കുകയും 5 എം.എൽ. രക്തം ശേഖരിക്കുകയും ചെയ്യും.
- ശേഖരിച്ച രക്തം സി-റിയാക്ട്ടിവ് പ്രോട്ടീൻ,സീറത്തിലെഅയർൺ, ട്ടോട്ടൽ അയർൺ ബൈൻഡിംഗ് കപ്പാസ്സിറ്റി, ഫെറിട്ടിൻ അളവ്കൺടുപിടിക്കാൻ ഉപയോഗിക്കുന്നതായിരിക്കും.

10. പ്രതീക്ഷിക്കുന്ന അപകട സാദ്ധ്യതകൾ -

ഈ പഠനത്തിന്റെ പ്രവർത്തനരീതി മൂലമുള്ള അപകട സാധ്യത വളരെ കുറവാണ്. മുറിവിൽനിന്നോ, തൊലിക്കടിയിൽ നിന്നോ ഉള്ള ചെറിയ രക്തസ്രാവം, വീക്കം, ചെറിയ വേദന എന്നിവയാണ് അപകട സാധ്യതകൾ. ഇവ സൂചിവച്ച സ്ഥലത്തിൽ ചെറിയ സമ്മർദ്ദം ചെലുത്തിയാൽ സാധുകരിക്കപ്പെടും.

11. ഈ പഠനത്തിൽ പങ്കെടുക്കുന്നതുകൊണ്ട് എനിക്ക് എന്തെങ്കിലും ഉപയോഗമുണ്ടോ ?

നിങ്ങൾക്ക് നേരിട്ട് ഒരു ഉപയോഗവും ഇല്ല.

12. ഞാൻ ഈ പഠനത്തിൽ പങ്കെടുക്കുന്ന വിവരം രഹസ്യമായി വയ്ക്കുമോ ?

നിങ്ങളിൽ നിന്ന് ശേഖരിക്കുന്ന എല്ലാ വിവരങ്ങളും രഹസ്യമായി വയ്ക്കുന്നതായിരിക്കും നിങ്ങളെ പറ്റിയുള്ള വിവരങ്ങൾ ആരോടും വെളിപ്പെടുത്തുന്നതായിരിക്കില്ല. ഭാവിയിൽ ഈ പഠനം ശാസ്ത്രലേഖനമായി പ്രസിദ്ധീകരിക്കുമ്പോൾ നിങ്ങളുടെ പേരിനു പകരം കോഡ് ആണ് ഉപയോഗിക്കുക.

13. എന്നെ എന്തുകൊണ്ട് ഈ പഠനത്തിൽ ഉൾപ്പെടുത്തി ?

നിങ്ങൾക്ക് പെരിഡോന്റൈറ്റിസ്എന്ന അസുഖം ഉള്ളതുകൊണ്ട് നിങ്ങളെ ഈ പഠനത്തിൽ ഉൾപെടുത്തിയിരിക്കുന്നു.

14. എത്ര ആളുകൾ ഈ പഠനത്തിൽ ഉൾപ്പെടുന്നു. 60

15. നഷ്ടപരിഹാര ഉടമ്പടി

പഠനവിധേയമായി ഏതെങ്കിലും തരത്തിൽ രോഗം സങ്കീർണ്ണമായാൽ രോഗിയെ ഈ സ്ഥാപനത്തിൽ വിദഗ്ദചികിത്സയ്ക്ക് വിധേയനാക്കുന്നതാണ്.

16. ഏതെങ്കിലും വിധത്തിൽ വേതനം ലഭിക്കുമോ : ഇല്ല

17. എപ്പോൾ വേണമെങ്കിലും എനിക്ക് ഈ പഠനത്തിൽ നിന്ന് പിൻമാറാമോ

ഈ പഠനത്തിൽ പങ്കെടുക്കണമോ വേണ്ടയോ എന്നു തീരുമാനിക്കാനുള്ള പൂർണ്ണ അവകാശം നിങ്ങൾക്കുണ്ട്. നിങ്ങൾ ഇതിൽ പങ്കെടുക്കുവാൻ തീരുമാനിച്ചാൽ ഒരു സമ്മതപത്രത്തിൽ ഒപ്പിട്ട് നൽകേണ്ടതുണ്ട്. ഈ പഠനത്തിൽ നിന്ന് ഏതു സമയവും പിൻവാങ്ങാനുള്ള സ്വാതന്ത്ര്യവും നിങ്ങൾക്കുണ്ട്. ഇത് നിങ്ങളുടെ മറ്റു ചികിത്സകളെ യാതൊരു വിധത്തിലും ബാധിക്കുന്നതായിരിക്കില്ല.

18. ഈ ഗവേഷണത്തിന്റെ ഫലമായി പുതിയ എന്തെങ്കിലും കണ്ടെത്തലുകളുണ്ടെങ്കിൽ അത് എന്നെ അറിയിക്കുമോ ?

ഈ പഠനത്തിന്റെ കണ്ടെത്തലുകൾ പഠന അവസാനം നിങ്ങളെ അറിയിക്കുന്നതായിരിക്കും.

19. ഈ പഠനത്തിന്റെ സമയ ദൈർഘ്യം എത്രയാണ് ?

ഏകദേശം 6 മാസം

20. ഇതുമായിബന്ധപ്പെടാത്ത ഏതെങ്കിലും വിവരങ്ങൾ ആവശ്യമുണ്ടോ - ഇല്ല

21. കൂടുതൽ വിവരങ്ങൾക്കായി താഴെ പറയുന്നവരെ നിങ്ങൾക്ക് ബന്ധപ്പെടാവുന്നതാണ്.

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ശ്രീ മൂകാംബിക ഇൻസ്റ്റിറ്റ്യൂട്ട് ഓഫ് ഡെന്റൽ സയൻസ്,
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സ്ഥലം:

തീയതി :

Annexure 3

பங்கேற்பாளர்களுக்கு ஆய்வினைக் குறித்த தகவல்

அன்பார்ந்த பங்கேற்பாளர்களே,

இந்த ஆராய்ச்சியில் தங்களை ஈடுபடுத்திக்கொள்ள மிகுந்த ஆர்வத்துடன் முழுமனதுடன் கலந்துகொள்ள வந்த வரவேற்பாளர்களை வரவேற்கிறேன். நீங்கள் இந்த ஆராய்ச்சியில்பங்கெடுத்துக்கொள்வதற்கு முன் இந்த ஆராய்ச்சி எதற்காக நடத்தப்படுகிறது என்பதை தெளிவாக புரிந்துகொள்ளவேண்டும். உங்களுக்கு தேவையான அனைத்து விபரங்களும் கீழே கொடுக்கப்பட்டுள்ளது. இந்த ஆராய்ச்சியின் மூலம் ஏற்படும் நன்மைகள், ஏதேனும் ஆபத்துகள் மற்றும் அதற்காக மற்றும் எவ்வாறு இந்த ஆராய்ச்சி மேற்கொள்ளப்படும் முறைகளையும் தெரிவிக்கப்பட்டுள்ளது. இதில் கொடுக்கப்பட்டுள்ள விபரங்களை தெளிவாக படித்து புரிந்து கொள்ளவேண்டும். நீங்கள் ஆராய்ச்சியில் பங்கேற்பாளர்களாக ஒப்புதல் வழங்குவதற்கு முன்பு உங்களுக்கு ஏற்படும் அறிவியல் சார்ந்த சந்தேகங்கள் மற்றும் ஆராய்ச்சி சம்பந்தப்பட்ட சந்தேகங்கள் அனைத்தும் இந்த ஆராய்ச்சியின் எந்த காலகட்டத்திலும் நீங்கள் படிவத்தில் குறிப்பிட்ட நபரிடம் கேட்டு தெளிவுபடுத்திக்கொள்ளலாம்.

1. தலைமை ஆய்வாளர்:

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2. வழிகாட்டியின் பெயர் :

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3. இணை வழிகாட்டியின் பெயர்:

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மருத்துவர். பீனா உண்ணிக்கிருஷ்ணன்
இணை பேராசிரியர், பொது நல மருத்துவத்துறை,
ஸ்ரீ மூகாம்பிகா மருத்துவக் கல்லூரி,குலசேகரம்-629161
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4. கல்லூரியின் பெயர் :

ஸ்ரீ மூகாம்பிகா பல் மருத்துவக்கல்லூரி மற்றும் மருத்துவமனை,
வி.பி.எம். காம்பிளக்ஸ்,
படநிலம், குலசேகரம்: 629 161.

5. ஆராய்ச்சியின் தலைப்பு

ஸ்கெயிலிங் & றூட்பிளானிங்கிற்கு முன்பும் பின்பும் பெரியோடோன்டைற்றிஸ் நோயாளிகளிலும், ஹீமோடயாலிஸிஸ் செய்கின்ற பெரியோடோன்டைட்டிஸ் நோயாளிகளில் சி-ரியாக்டிவ் புரோட்டின் மற்றும் இரும்பு குறியீடுகளில்ஏற்படும் அளவின் வித்தியாசம்.

6. குறிக்கோள்:

1995 முதல் 2014 வரையிலான வெளியிடப்பட்டுள்ளவிஞ்ஞான புத்தகங்களின் இருந்து தெர்ந்தெடுக்கப்பட்ட விவரங்கள்.

7. ஆய்வின் நோக்கம் மற்றும் குறிக்கோள்கள்

இந்த **ஆய்வின் குறிக்கோள்,**ஸ்கெயிலிங் & றூட்பிளானிங்கிற்கு முன்பும் பின்பும் பெரியோடோன்டைற்றிஸ் நோயாளிகளிலும், ஹீமோடயாலிஸிஸ் செய்கின்ற பெரியோடோன்டைட்டிஸ் நோயாளிகளில் சி-ரியாக்டிவ் புரோட்டின் மற்றும் இரும்பு குறியீடுகளில்ஏற்படும் அளவின் வித்தியாசம்.

8. ஆராய்ச்சியை அறிவியல் ரீதியாக உறுதிபடுத்தல்:

ஸ்கெயிலிங் & றூட் பிளானிங் சிகிட்சை பாக்டீரியாக்களின் அளவை குறைக்கும்அதன் மூலம்ஸீரமில் சி-ரியாக்டிவ் புரோட்டின்அளவை குறைக்கவும்,இரும்பு சத்து, மொத்த இரும்புசத்து பிணைதிறன் மற்றும் ∴பரிட்டின் ஆகியவற்றை அதிகபடுத்தவும் அதன்வழியாக பெரியோடான்டைட்டிஸின் ஆரோக்கியநிலை மெச்சப்படுத்தவும் செய்கிறது.

9. செய்முறை :

- தங்கள் இந்த ஆராய்ச்சியில் ஈடுபட்டால், தங்களுக்கென்று ஒரு எண் வழங்கப்படும்
- உங்களின் முழு விவரங்கள் அடங்கின கேஸ் சார்ட் எழுதப்படும். தேவையென்றால் பரிசோதனை நடத்தப்படும். தேவைக்கேற்ப படங்களும் எடுக்கப்படும்.
- பெரியோடான்டைட்டிஸ் பரிசோதனை வேளையில் 5ml இரத்தம் எடுக்கப்படும்.
- ஊசி மூலம் இரத்தம் எடுக்கப்படும். இது இரத்தத்தில் உள்ளசி-ரியாக்டிவ் புரோட்டின், இரும்பு சத்து, மொத்த இரும்புசத்து பிணைதிறன் மற்றும் ∴பரிட்டின் என்ற வஸ்துவின் அளவையும் பரிசோதிப்பதற்கு உபயோகப்படுத்தப்படும்.
- இரத்தம் எடுப்பது காலை 8 மணிக்கும் 10 மணிக்கும் இடையில் எடுக்கப்படும்.
- அதன்பிறகு ஸ்கெயிலிங் & றூட் ப்ளானிங் என்ற சிகிட்சை செய்வதாகும்.
- ஒரு மாதத்திற்கு பிறகு உங்களுடைய பெரியோடோன்டிஷத்தை பரிசோதிக்க 5ml இரத்தம் எடுக்கப்படும்.

10. எதிர்பார்க்கும் பக்கவிளைவுகள்

இச்செயல்பாட்டின் விளைவுகள் சிறிய அளவிலான பிரச்சனைகளையே ஏற்படுத்துகிறது. புண் அல்லது தோலுக்கடியில் சிறிய அளவு இரத்தம் வெளிப்பட்டு அது வீக்கமாக மாறி வலியை ஏற்படுத்தலாம். ஊசி போட்ட பிற்பாடு அந்த இடத்தை அழுத்தமாக தேய்த்து விட்டால் இப்பிரச்சனை வராமல் தடுக்கலாம்.

11. ஆராய்ச்சியில் ஈடுபடுவர்களுக்கு எதிர்பார்க்கும் நன்மைகள் ?

நேரடி பயன் எதுவும் இல்லை. இருப்பினும் தங்களுக்கு இரத்த பரிசோதனைக்கு உண்டாகும் செலவினை நாங்கள் ஏற்றுக்கொள்கிறோம்.

12.தங்களைப் பற்றிய விவரங்ள் அனைத்தும் நம்பிக்கைக்கு உரியதாக பாதுகாக்கப்படும் ?

இந்த ஆய்வின்மூலம் நோயாளிகளுக்கு அவர்களின் நோய் குறித்த முன்கணிப்பை புரிந்துகொள்ள முடியும்.

13.எதற்காக நான் இந்த ஆராய்ச்சிக்கு தேர்ந்தெடுக்கப்பட்டேன்?

உங்களுக்கு பெரிடோன்டைட்டிஸ் நோய் இருப்பதினால் உங்களை இந்த ஆய்வுக்கு தேர்ந்தெடுக்கப்பட்டுள்ளது.

14. எத்தனை பேர் இவ்வாராய்ச்சியில் பங்கு கொள்வார்கள் ? 60

15. இந்த ஆராய்ச்சியில் ஏதேனும் தீங்கு ஏற்பட்டால் அது எவ்வாறு ஈடு செய்யப்படும்?

இந்த ஆராய்ச்சியினால் தங்களுக்கு நேரும் எந்தவித விபரீதங்களுக்கும் எங்கள் முதன்மை ஆராய்ச்சியாளர் அதன் முழு மருத்துவ செலவினை ஏற்றுக்கொள்வார்

16. இந்த ஆராய்ச்சியில் பங்கேற்பதற்காக கொடுக்கப்படும் தொகை?இல்லை

17. நான் ஆராய்ச்சியின் இடையில் விலகிக் கொள்ளலாமா?

தங்களுக்கு இந்த ஆராய்ச்சியில் பங்கு கொள்ளவும் விலகவும் முழு சுதந்திரம் உண்டு. ஆராய்ச்சியில் பங்குகொள்வதாலும், இடையில் விலகுவதாலும் தங்கள் சிகிட்சைக்குஎந்தவித பாதிப்பும் ஏற்படாது.

18. ஏதேனும் புதிய தகவல்களோ கண்டுபிடிப்புகளோ இவ்வாரய்ச்சியில் கண்டுபிடிக்கப் பட்டால் என்னிடம் விவரம் தெரிவிக்கப்படுமா?ஆம்

19. எவ்வளவு காலம் இந்த ஆராய்ச்சியில் ஈடுபடநேரியும் ?ஆறுமாதம்

20. இவ்வாராய்ச்சியைப் பற்றிய இதர தகவல்கள் ?இல்லை

21. ஏதேனும் விவரம் வேண்டுமென்றால் யாரை அணுக வேண்டும் ?

விவரம் அறிந்துகொள்ள என்னை தாராளமாக தொடர்புகொள்ளலாம்

டாக்டர். சீதள் மேனோன். வி
முதுகலை மாணவவி
பெரியோடான்டிக்ஸ் துறை
ஸ்ரீ மூகாம்பிகா பல் மருத்துவக்கல்லூரி மற்றும் மருத்துவமனை,
குலசேகரம்-629 161
9445678206
Sheethel.menon@gmail.com

இடம் :
தேதி :

முதன்மை ஆராய்ச்சியாளரின் கையொப்பம்

Annexure 4

PARTICIPANTS CONSENT FORM

The details of the study have been explained to me in writing and the details have been fully explained to me. I am aware that the results of the study may not be directly beneficial to me but will help in the advancement of medical sciences. I confirm that I have understood the study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). I have been given an information sheet giving details of the study. I fully consent to participate in the study titled‘Effect of nonsurgical periodontal therapy on C-reactive protein and iron indices in haemodialysis patients with chronic periodontitis’

Serial no / Reference no:

Name of the participant:

Address of the participant:

Contact number of the participant:

Signature of the participant:

Witnesses:

1.
2.

Date:

Place

Annexure 4

സമ്മതപത്രം

ഈ പഠനത്തെ പറ്റിയുള്ള എല്ലാ കാര്യങ്ങളും എനിക്ക് പറഞ്ഞ് മനസ്സിലാക്കി തരികയും അതിന്റെ ഒരു പകർപ്പ് എനിക്കു നൽകുകയും ചെയ്തിട്ടുണ്ട്. ഈ പഠനം ഗവേഷണത്തിനായി ഉള്ളതാണെന്നും എനിക്ക് ഇതിൽ നിന്ന് നേരിട്ട് ഒരു ഫലവും ഉണ്ടാകില്ലെന്നും ഞാൻ മനസ്സിലാക്കുന്നു. ഈ പഠനത്തിന്റെ രീതിയും ഉദ്ദേശവും എനിക്ക് മനസ്സിലാക്കി തന്നിട്ടുണ്ട്. അതു പോലെ എനിക്ക് സംശയങ്ങൾ ചോദിക്കാൻ അവസരങ്ങൾ ലഭിച്ചിട്ടുമുണ്ട്. ഇതിൽ പങ്കെടുക്കാനും പങ്കെടുക്കാതിരിക്കാനും ഉള്ള അവകാശം എനിക്കുണ്ടെന്നും അതുപോലെ പഠനത്തിന്റെ ഏതു ഘട്ടത്തിലും ഇതിൽ നിന്ന് പിൻവങ്ങാനുള്ള സ്വാതന്ത്ര്യവും എനിക്കുണ്ടെന്നും ഞാൻ മനസ്സിലാക്കുന്നു. ഈ പഠനത്തിൽ പങ്കെടുക്കുന്നതുകൊണ്ടോ, പങ്കെടുക്കാത്തതുകൊണ്ടോ എന്റെ മറ്റു ചികിത്സകളെ ബാധിക്കുന്നതല്ലെന്ന് ഞാൻ അിറയുന്നു. **"**സ്കെയ്ലിംഗ് & റൂട്ട് പ്ലേനിംഗ് ചികിത്സയ്ക്ക് മുൻപും ശേഷവും പെരിഡോന്റൈറ്റിസ് രോഗികളിലും, ഹീമോഡയാലിസിസ് ചെയ്യുന്ന പെരിഡോന്റൈറ്റിസ്രോഗികളിലും ഉണ്ടാകുന്ന സി-റിയാക്ട്ടിവ് പ്രോട്ടീനിലും അനീമിയാ- ലും വരുന്ന വ്യത്യാസം.**"** എന്ന ഗവേഷണത്തിൽ പങ്കെടുക്കുന്നതിനും ഇതിന്റെ ഫലങ്ങൾ ശാസ്ത്രലേഖനത്തിൽ പ്രസിദ്ധീകരിക്കുന്നതിനും എനിക്ക് സമ്മതമാണെന്ന് ഞാൻ ഇതിനാൽ അിറയിച്ചുകൊള്ളുന്നു.

സീരിയൽ നമ്പർ / റഫറൻസ് നമ്പർ :
പങ്കെടുക്കുന്ന ആളിന്റെ പേര് :
മേൽവിലാസം :
ഫോൺ നമ്പർ :

ഒപ്പ് / വിരലടയാളം

സാക്ഷി :

സ്ഥലം :
തീയതി

Annexure 4

ஒப்புதல் படிவம்

இந்த ஆராய்ச்சியின் தகவல்கள் அனைத்தும் என்னிடம் தெளிவாக எழுத்துமூலம் விளக்கப்பட்டுள்ளது. இந்த ஆராய்ச்சியின் முடிவுகள் எனக்கு நேரடியாக பயன்பராவிட்டாலும் மருத்துவத்துறையின் முன்னேற்றத்திற்கு பயன்படும் என்பதை அறிவேன். இவ்வாராய்சியைப் பற்றி நான் தெளிவாக புரிந்துக் கொண்டுள்ளேன். நான் தானாக முன்வந்து இதில் பங்குப் பெறுகிறேன். என்பதை அறிவேன். இதிலிருந்து எந்த நேரமும் எக்காரணமும் கூறாமல் வந்தாலும் இந்த மருத்துவமனையில் எனக்கு கிடைக்கும் மருத்துவ உதவி எவ்விதத்திலும் பாதிக்கப்படாது என்பதையும் அறிவேன். இவ்வாராய்ச்சியின் மூலம் வரும் முடிவுகள் மற்றும் தகவல்களை அறிவியல்துறையின் பயன்பாடுகளுக்கு (மட்டுமே) உபயோகப்பட்டிக்கொள்ள சம்மதிக்கிறேன். எனக்கு இவ்வாராய்ச்சியைப் பற்றிய விரிவான தகவல்கள் அடங்கிய படிவம் தரப்பட்டுள்ளது.

நான் "ஸ்கெயிலிங் & றூட்பிளானிங்கிற்கு முன்பும் பின்பும் பெரியோடோன்டைற்றிஸ் நோயாளிகளிலும், ஹீமோடயாலிஸிஸ் செய்கின்ற பெரியோடோன்டைட்டிஸ் நோயாளிகளில் சி-ரியாக்டிவ் புரோட்டின் மற்றும் இரும்பு குறியீடுகளில்ஏற்படும் அளவின் வித்தியாசம்" என்கிற இவ்வாராய்ச்சியில் பங்கேற்க முழுமனதுடன் சம்மதிக்கிறேன்.

குறிப்பு எண் :

பெயர் :

முகவரி :

தொலைபேசி எண் : கையொப்பம்

சாட்சி 1

2

Annexure 5

SREEMOOKAMBIKA INSTITUTE OF DENTAL SCIENCES
CASE RECORD FORM

Title of the short study: 'Effect of nonsurgical periodontal therapy on C-reactive protein and iron indices in haemodialysis patients with chronic periodontitis'

NAME:	DATE:	PHONE NUMBER:
AGE:	SEX:	ADDRESS:

INITIAL RECORDING (T_0)

Smoking status:

Weight: Height: BMI:

Blood Pressure Systolic: Diastolic:

Renal and Hematological Parameters

CRP	
Serum Iron	
TIBC	
Transferrin saturation	
Serum Ferritin	
Hb	
ESR	
Albumin	
Creatinine	
GFR	

Medical history

a) If patient is under haemodialysis:

Dialysis duration:

Annexure 5

Clinical Parameters

Number of teeth:

PLAQUE INDEX

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Plaque Index Score:

GINGIVAL INDEX

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Gingival Index Score:

Bleeding on Probing:

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Annexure 5

PROBING POCKET DEPTH

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

GINGIVAL RECESSION/GINGIVAL ENLARGEMENT

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

CLINICAL ATTACHMENT LEVEL

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Treatment done:-

Signature of Investigator

Annexure 5

30 DAYS POST OPERATIVE RECORDING(T_1)

Clinical Parameters

Number of teeth:

PLAQUE INDEX

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Plaque Index Score:

GINGIVAL INDEX

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Gingival Index Score:

Bleeding on Probing:

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Annexure 5

PROBING POCKET DEPTH

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

GINGIVAL RECESSION/GINGIVAL ENLARGEMENT

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

CLINICAL ATTACHMENT LEVEL

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Treatment done:-

Signature of Investigator

Annexure 5

90 DAYS POST OPERATIVE RECORDING(T_2)

Renal and Hematological Parameters

CRP	
Serum Iron	
TIBC	
Transferrin saturation	
Serum Ferritin	
Hb	
ESR	
Albumin	
Creatinine	
GFR	

Clinical Parameters

Number of teeth:

PLAQUE INDEX

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Plaque Index Score:

Annexure 5

GINGIVAL INDEX

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Gingival Index Score:

Bleeding on Probing:

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Annexure 5

PROBING POCKET DEPTH

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

GINGIVAL RECESSION/GINGIVAL ENLARGEMENT

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

CLINICAL ATTACHMENT LEVEL

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28

48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

Treatment done:-

Signature of Investigator

Annexure 6

Group 1(Haemodialysis<1 year)

Sl. No	Name	Age	Sex	BMI(kg/m^2)	BP(Sys/Dia)	No of teeth present
1	Paulraj	52	M	19.921	`132/96	30
2	Sunder	55	M	21.60	140/96	24
3	Saraswathy	59	F	2573	136/96	22
4	Ajith	41	M	26.5	144/100	27
5	Vikraman	62	M	20.55	138/96	31
6	Kala	34	F	22.07	130/82	29
7	Sivalingam	54	M	25.8	132/90	30
8	Nainar	65	M	20.31	138/100	28
9	Rathnakumar	50	M	23.80	132/92	23
10	Fathima	35	F	20.56	136/88	32
11	Margaret	60	F	24.69	138/96	22
12	George	53	M	20.95	132/90	30
13	Thankapandi	61	M	22.4	138/90	29
14	Dasan	51	M	24.30	130/94	22
15	Balakrishnan	58	M	20.31	138/96	31
16	Dennison	45	M	20.51	136/94	24
17	Gabriel	45	M	23.23	136/96	32
18	Suresh	36	M	16.73	134/96	32
19	John	44	M	18.59	136/92	26
20	George	63	M	26.56	140/96	23

Annexure 6

Group 2(Haemodialysis>1 year)

Sl. No	Name	Age	Sex	BMI(kg/m^2)	BP(Sys/Dia)	No of teeth present
1	Ponnappan	67	M	24.63	138/94	23
2	Masilamani	65	M	22.05	136/96	26
3	Devadhas	54	M	24.38	130/90	29
4	Vijayakumar	30	M	19.87	136/94	32
5	Nazeema	46	F	24.34	140/96	27
6	Vijayakumar	50	M	23.11	138/96	28
7	Mani	58	M	22.36	138/94	23
8	Natarajan	64	M	24.25	136/90	23
9	Justin	36	M	22.40	136/94	28
10	Brightdas	50	M	22.86	138/92	30
11	Aruldas	43	M	22.65	140/96	30
12	Kanakaraj	45	M	22.86	132/92	31
13	Subraman	61	M	23.23	136/94	32
14	Reymond	42	M	21.707	138/90	32
15	Merlin	28	F	21.30	130/90	30
16	Baby Girija	56	F	23.92	140/98	22
17	Thapamani	50	M	26.61	136/86	24
18	Ganeshan	64	M	23.04	134/90	20
19	Albin Jose	32	M	24.5	140/96	29
20	Marianesan	57	M	24.6	138/94	32

Annexure 6

Group 3(Chronic Periodontitis)

Sl. No	Name	Age	Sex	BMI(kg/m^2)	BP(Sys/Dia)	No of teeth present
1	Sandhya	28	F	21.35	120/84	30
2	Kaladevi	49	F	20.84	120/82	28
3	Ganesh	33	M	21.88	120/80	29
4	Nalini	41	F	24.2	110/70	27
5	Syamala	46	F	19.69	110/80	30
6	Ratheesh	44	M	26.3	122/82	25
7	Marybai	40	F	21.6	122/80	22
8	Annamma	55	F	25.51	126/84	28
9	Gopi	54	M	26.67	120/80	30
10	Shikha	35	F	24.91	120/80	29
11	Anandaroopan	27	M	23.91	112/80	31
12	Mary	40	F	23.62	110/82	26
13	Sreekumar	55	M	24.44	110/70	31
14	Pushpakaran	51	M	25.3	130/86	21
15	Suji	33	F	26.34	120/80	23
16	Vijila	38	F	24.69	126/82	30
17	Lekshmi	40	F	21.48	120/80	30
18	Saritha	29	F	18.28	110/86	31
19	Jayanthi	43	F	19.28	120/80	31
20	Jerald	46	M	19.72	120/80	31

Annexure 6

Group 1(Haemodialysis<1 year)

Sl NO	Name	Plaque index			Bleeding on Probing			Gingival index		
		T0	T1	T2	T0	T1	T2	T0	T1	T2
1	Paulraj	1.35	1.33	1.31	32.7	23.8	21.1	1.41	1.36	1.31
2	Sunder	2.4	1.70	1.67	29.1	22.9	20.1	1.42	1.45	1.34
3	Saraswathy	1.28	0.63	1.4	34.09	28.78	21.9	1.44	1.17	1.16
4	Ajith	0.66	0.57	0.55	29	22	24.07	1.34	1.03	1.02
5	Vikraman	2.32	1.58	1.48	36	28.4	26.3	1.56	1.41	1.33
6	Kala	2.44	1.49	1.70	43.6	38.5	28.7	1.72	1.52	1.39
7	Sivalingam	1.95	1.66	1.67	22.7	18.8	14.4	1.35	1.28	1.35
8	Nainar	1.9	1.64	1.58	27	28.4	27.3	1.71	1.36	1.28
9	Rathnakumar	2.54	1.60	1.19	73.9	42.7	24.6	2.53	1.48	1.33
10	Fathima	1.65	1.28	1.20	22.91	16.1	13.5	1.41	1.179	1.26
11	Margaret	2.18	1.43	1.54	36.2	23.4	18.8	1.59	1.38	1.34
12	George	1.63	1.4	1.4	36.6	32	26.1	1.5	1.41	1.32
13	Thankapandi	1.80	1.40	1.34	32.1	30.4	17.2	1.46	1.39	1.34
14	Dasan	2.19	1.45	1.42	38.63	29.5	24.2	1.82	1.36	1.375
15	Balakrishnan	1.21	0.93	0.87	13.4	13.4	10.7	1.26	1.29	1.25
16	Dennison	1.32	0.65	0.781	29.16	25.6	22.9	2	1.3	1.39
17	Gabriel	1.44	1.40	1.38	36.9	25.5	19.79	1.53	1.46	1.35
18	Suresh	1.52	1.48	1.46	34	33	33.3	1.63	1.57	1.37
19	John	2.3	1.49	1.43	41.2	28.2	21.1	1.63	1.41	1.35
20	George	1.59	1.63	1.47	34.05	28.2	24.6	1.48	1.39	1.31

Annexure 6

Sl NO	Name	Probing pocket depth			Clinical Attachment Level		
		T0	T1	T2	T0	T1	T2
1	Paulraj	2.86	2.81	2.72	3.2	3.02	3.1
2	Sunder	2.57	2.62	2.34	3.95	3.48	3.46
3	Saraswathy	3.18	2.75	2.62	3.24	3.21	3.09
4	Ajith	2.95	2.54	2.50	3.19	2.79	2.73
5	Vikraman	2.74	2.66	2.51	3.94	3.59	3.39
6	Kala	2.97	2.85	2.68	3.02	2.89	2.73
7	Sivalingam	2.73	2.67	2.64	2.79	2.73	2.71
8	Nainar	3.34	2.77	2.69	3.39	2.81	2.72
9	Rathnakumar	3.54	3.39	3.23	3.97	3.72	3.52
10	Fathima	2.60	2.16	2.46	2.66	2.21	2.48
11	Margaret	2.45	2.48	2.42	2.75	2.80	2.77
12	George	3.47	3.49	3.23	3.73	3.74	3.39
13	Thankapandi	2.31	2.63	2.49	2.45	2.72	2.56
14	Dasan	2.91	2.43	2.52	5.08	4.96	4.67
15	Balakrishnan	3.18	3.23	3.13	3.25	3.31	3.22
16	Dennison	3.03	3.12	3.12	3.31	3.36	3.36
17	Gabriel	3.09	2.72	2.63	3.43	3.06	2.93
18	Suresh	3.16	2.65	2.63	3.05	2.99	2.91
19	John	2.7	2.63	2.53	3.53	3.371	3.23
20	George	3.09	3.06	2.9	3.76	3.71	3.52

Annexure 6

Group 2(Haemodialysis>1 year)

Sl NO	Name	Plaque index			Bleeding on Probing			Gingival index		
		T0	T1	T2	T0	T1	T2	T0	T1	T2
1	Ponnappan	2.02	1.58	1.92	60.14	45.6	42	1.75	1.59	1.69
2	Masilamani	1.4	0.99	1.48	35.25	27.5	26.9	1.52	1.31	1.35
3	Devadhas	1.74	1.5	1.64	43.6	39.08	33.9	1.72	1.46	1.44
4	Vijayakumar	2.18	1.28	1.72	31.25	22.3	15.6	1.5	1.35	1.28
5	Nazeema	1.5	0.93	0.86	19.1	16.04	13.5	1.26	1.12	1.20
6	Vijayakumar	1.58	1.35	1.57	43.4	36.30	25	2.18	1.46	1.26
7	Mani	1.55	1.52	1.36	39.13	28.9	23.9	1.76	1.41	1.33
8	Natarajan	1.8	1.61	1.57	46.37	31.8	28.9	1.70	1.43	1.43
9	Justin	1.75	1.4	0.84	30.95	25	17.2	1.41	1.34	1.13
10	Brightdas	1.52	1.41	1.47	21.6	14.4	11.6	1.28	1.23	1.1
11	Aruldas	1.98	1.43	1.41	23.8	21.6	18.3	1.37	1.3	1.31
12	Kanakaraj	2.09	1.54	1.52	47.3	37.6	34.9	2.25	1.58	1.41
13	Subraman	2.05	1.54	1.44	38.5	33.3	28.12	1.56	1.51	1.39
14	Reymond	1.32	1.34	1.37	23.95	21.3	25.7	1.27	1.28	1.23
15	Merlin	1.68	1.41	1.38	32.7	29.4	20.5	1.48	1.39	1.35
16	Baby Girija	1.29	0.81	0.87	12.8	7	9	1.60	1.29	1.34
17	Thapamani	2.07	1.61	1.68	43.05	31.9	27.08	1.62	1.44	1.45
18	Ganeshan	1.81	1.4	1.53	43.3	32.5	30	1.47	1.43	1.36
19	Albin Jose	1.81	1.59	1.32	9.7	8	7	1.13	1.17	1.19
20	Marianesan	1.81	1.32	1.315	41.6	31.7	27.08	1.89	1.37	1.35

Annexure 6

Sl NO	Name	Probing pocket depth			Clinical Attachment Level		
		T0	T1	T2	T0	T1	T2
1	Ponnappan	2.69	2.64	2.54	3.05	2.92	2.80
2	Masilamani	3.43	3.28	3.44	3.52	3.35	3.51
3	Devadhas	3.60	2.97	3.32	5.09	4.67	4.56
4	Vijayakumar	2.93	2.80	2.56	3	2.86	2.62
5	Nazeema	2.70	2.33	2.31	2.77	2.40	2.34
6	Vijayakumar	3.35	3.22	3.13	3.71	3.63	3.45
7	Mani	2.76	2.60	2.51	2.971	2.68	2.57
8	Natarajan	4.31	4.20	4.26	5.5	5.20	5.35
9	Justin	3.04	2.91	2.87	3.065	2.92	2.88
10	Brightdas	2.56	2.3	2.3	2.62	2.43	2.41
11	Aruldas	2.66	2.61	2.48	2.78	2.72	2.57
12	Kanakaraj	2.67	2.65	2.63	3.33	3.33	3.2
13	Subraman	2.65	2.78	2.53	2.71	2.74	2.58
14	Reymond	2.92	2.70	2.69	3.05	2.84	2.80
15	Merlin	3.02	3.05	2.94	3.08	3.1	2.99
16	Baby Girija	2.90	2.70	2.62	2.96	2.78	2.70
17	Thapamani	3.28	3.19	3.04	3.95	3.86	3.59
18	Ganeshan	2.63	2.35	2.3	3.62	3.35	3.25
19	Albin Jose	2.67	2.44	2.43	2.68	2.45	2.44
20	Marianesan	2.85	2.76	2.73	3.71	3.02	2.94

Annexure 6

Group 3(Chronic Periodontitis)

Sl NO	Name	Plaque index			Bleeding on Probing			Gingival index		
		T0	T1	T2	T0	T1	T2	T0	T1	T2
1	Sandhya	2.43	1.92	1.6	84.4	82	33.8	2.6	2.12	1.54
2	Kaladevi	1.92	1.52	1.4	54	35.1	35.7	2.10	1.47	1.57
3	Ganesh	1.86	1.73	1.54	52.8	37.9	29.3	2.10	1.64	1.41
4	Nalini	1.52	1.38	1.38	37.03	27.7	24.6	1.47	1.38	1.34
5	Syamala	1.70	1.42	1.62	30	23.8	21.6	1.47	1.27	1.36
6	Ratheesh	1.86	1.38	1.4	45.3	42.6	19.3	1.72	1.49	1.3
7	Marybai	2.55	1.48	1.28	62.8	40.1	24.2	1.93	1.51	1.29
8	Annamma	1.72	0.99	1.37	30.95	21.4	15.4	1.44	1.26	1.16
9	Gopi	2.30	1.55	1.33	47.7	40	26.6	2.33	1.56	1.4
10	Shikha	1.80	1.52	1.5	55.1	26.4	28.7	1.56	1.32	0.91
11	Anandaroopan	1.70	1.65	1.4	63.4	46.7	23.11	1.88	1.70	1.37
12	Mary	1.42	1.27	0.98	41.02	23	19.87	1.43	1.26	1.25
13	Sreekumar	1.19	1.45	1.37	31	32.2	22.5	1.18	1.45	1.29
14	Pushpakaran	1.65	1.54	0.92	36.5	24.6	11.9	1.53	1.32	1.23
15	Suji	1.62	0.93	1.02	35.5	21.7	18.8	1.54	1.34	1.32
16	Vijila	2.0	1.45	1.40	45	30.5	23.8	1.67	1.37	1.36
17	Lekshmi	1.21	1.5	1.5	48.3	33.3	23.3	1.7	1.38	1.3
18	Saritha	1.79	1.67	1.45	38.17	24.19	23.11	1.49	1.29	1.27
19	Jayanthi	1.71	1.68	1.516	56.9	44	24.7	1.73	1.53	1.5
20	Jerald	1.87	1.37	1.20	24.1	15.5	13.9	1.33	1.25	1.16

Annexure 6

Sl NO	Name	Probing pocket depth			Clinical Attachment Level		
		T0	T1	T2	T0	T1	T2
1	Sandhya	3.81	3.5	2.73	3.93	3.75	2.84
2	Kaladevi	2.92	2.67	2.58	2.95	2.75	2.63
3	Ganesh	2.85	2.71	2.84	3.04	2.88	3.04
4	Nalini	2.73	2.69	2.96	3.20	3.17	3.38
5	Syamala	2.93	2.78	2.55	3.14	3	2.75
6	Ratheesh	3.59	3.34	3.21	3.90	3.64	3.45
7	Marybai	4.06	3.75	3.62	4.88	4.50	4.22
8	Annamma	2.69	2.82	2.61	2.73	2.87	2.67
9	Gopi	2.87	2.87	2.86	3.16	3.16	3.11
10	Shikha	2.68	2.74	2.70	2.86	2.90	2.85
11	Anandaroopan	3.74	3.60	3.49	3.80	3.67	3.55
12	Mary	2.70	2.69	2.76	2.78	2.77	2.84
13	Sreekumar	2.94	3.01	2.81	3.24	3.18	3.04
14	Pushpakaran	3.84	3.57	3.06	4.31	4.04	3.53
15	Suji	2.26	2.143	2.04	2.39	2.154	2.05
16	Vijila	3.52	3.48	3.32	3.7	3.73	3.47
17	Lekshmi	3.31	3.21	3.08	3.62	3.53	3.38
18	Saritha	2.63	2.5	2.49	2.74	2.58	2.5
19	Jayanthi	3.59	3.49	3.37	4.04	3.94	3.80
20	Jerald	2.48	2.5	2.5	3.01	3	2.96

Annexure 6

Group 1(Haemodialysis<1 year)

Sl NO	Name	CRP(T0) (mg/L)	CRP(T2) (mg/L)	Serum iron(T0) (µg/dl)	Serum iron(T2) (µg/dl)	Serum TIBC(T0) (µg/dl)	Serum TIBC(T2) (µg/dl)	TSAT(T0) %	TSAT(T2) %
1	Paulraj	2.1	1	29.5	59	154	212	19.15	27.83
2	Sunder	4	3.7	31.9	40	215.7	225	14.78	17.77
3	Saraswathy	3.1	3	27.6	30	177.2	184.2	15.57	16.28
4	Ajith	2	3.1	53.3	49	125.3	113.2	42.53	43.28
5	Vikraman	4.8	5.6	31.4	46.6	177.6	189.4	17.68	24.60
6	Kala	1.6	9.9	25.4	41.5	130.9	250.2	19.40	16.58
7	Sivalingam	3.2	1.2	35.6	33.5	198.7	221.6	17.91	15.11
8	Nainar	10	7	30.5	66.3	188.5	220.4	16.18	30.08
9	Rathnakumar	14	10	40.5	51	201	231.2	20.1	22.05
10	Fathima	12	7	54.3	64	165.1	189.5	32.88	33.77
11	Margaret	14	10	45.1	41.4	261.5	251.3	17.24	16.47
12	George	3.8	2.4	36.8	50.2	198.4	218.7	18.54	22.95
13	Thankapandi	4.6	4.5	40.2	45.6	210.2	223.5	19.12	20.40
14	Dasan	5.2	6	32.5	29	169.1	193	19.21	15.02
15	Balakrishnan	12	9	30.1	35.2	215.1	235	13.99	14.97
16	Dennison	7	5	46.7	72.9	174.3	210	26.79	34.71
17	Gabriel	5.5	2.8	49.8	51	182.3	187	27.31	27.27
18	Suresh	12.3	10.5	42.7	42	251.1	259	17	16.21
19	John	25	21	35.8	37.4	126.1	131.3	28.39	28.48
20	George	15	19	50.1	49.8	136.1	134.5	36.81	37.02

Annexure 6

Sl. No	Name	Serum ferritin (T0) (ng/mL)	Serum ferritin (T2) (ng/mL)	Hb(T0) (gm/dL)	Hb(T2) (gm/dL)	ESR(T0) (mm/Hr)	ESR(T2) (mm/Hr)	Serum albumin (T0) (gm/dL)	Serum albumin (T2) (gm/dL)
1	Paulraj	471.9	444	8.1	9	52	49	3.8	3.9
2	Sunder	320.7	147.5	7.5	10.2	112	90	4.1	3.8
3	Saraswathy	83	69	9.4	8.3	79	84	3.7	4
4	Ajith	230	267	7.1	8.7	44	53	3.5	3.8
5	Vikraman	306.1	864.1	8	9	90	69	3.7	3.9
6	Kala	355.8	429.2	6	5.6	92	72	3.8	4
7	Sivalingam	987.9	877	9.6	10.3	71	60	3.7	4.1
8	Nainar	930	906.1	6.4	7.8	40	30	4	3.8
9	Rathnakumar	987	856	10.9	11	81	77	3.8	4
10	Fathima	587.1	1126.3	9.6	8.5	61	37	3.5	3.6
11	Margaret	561	715.1	9.2	8.5	57	46	4.1	4.2
12	George	467	450	10.1	6.2	59	90	4.9	3.7
13	Thankapandi	368	334	12	12.7	66	45	4.3	3.9
14	Dasan	278	246	8	9.3	36	33	4.1	3.8
15	Balakrishnan	335	352	9.4	11	45	36	3.8	4.1
16	Dennison	304	286.9	10	13.2	55	37	4	4.2
17	Gabriel	413	401.6	9	9.8	60	76	3.8	4.1
18	Suresh	260	289	10	12	79	66	4.1	4.4
19	John	640	662.1	11.5	11	67	64	4	4.4
20	George	290	284	9	8.7	82	75	3.8	3.9

Annexure 6

Group 1(Haemodialysis<1 year)

Sl.No	Name	Serum creatinine (T0) (mg/dl)	Serum creatinine (T2) (mg/dl)	GFR(T0) (mL/ min/1.73m^2)	GFR(T2) (mL/min/ 1.73m^2)
1	Paulraj	11.1	10.1	5	6
2	Sunder	12.1	12.1	5	5
3	Saraswathy	5.1	7	10	7
4	Ajith	11.5	11	6	6
5	Vikraman	7	7.3	9	8
6	Kala	12	11.9	4	4
7	Sivalingam	6.4	7.4	10	9
8	Nainar	6.9	5.5	9	12
9	Rathnakumar	9.5	10	7	6
10	Fathima	9.3	8.5	6	6
11	Margaret	8.5	8.6	5	5
12	George	9.5	9.6	7	6
13	Thankapandi	6.5	8	10	8
14	Dasan	6.4	5	11	14
15	Balakrishnan	7.8	6.7	8	10
16	Dennison	8.2	11.6	8	5
17	Gabriel	9.1	9.5	7	7
18	Suresh	9.5	7.9	7	9
19	John	8.3	7.9	8	9
20	George	9.5	5.4	6	12

Annexure 6

Group 2(Haemodialysis>1 year)

Sl NO	Name	CRP(T0) (mg/L)	CRP(T2) (mg/L)	Serum iron(T0) (µg/dl)	Serum iron(T2) (µg/dl)	Serum TIBC(T0) (µg/dl)	Serum TIBC(T2) (µg/dl)	TSAT(T0) %	TSAT(T2) %
1	Ponnappan	4.8	4.8	42.4	46	189	195.6	19.15	23.51
2	Masilamani	5.1	18L	49.2	82	173	223	28.43	36.77
3	Devadhas	3.2	4	51	59	136	152	37.5	38.81
4	Vijayakumar	3.1	1.2	65	51	152	131.2	42.76	38.87
5	Nazeema	2.2	1.5	39.9	41.9	158	161.2	25.25	25.99
6	Vijayakumar	7	6.4	90	92	261.4	263.4	34.42	34.92
7	Mani	2.4	4	49	51.4	109.7	113.9	44.66	45.12
8	Natarajan	20	12	59	49.2	213.2	185.4	27.67	26.53
9	Justin	4.2	5	50.1	47	181.2	173.9	27.64	27.02
10	Brightdas	13	10	62	47	192	148	32.29	31.75
11	Aruldas	4.8	4	49.3	49	170.3	176.8	28.94	27.71
12	Kanakaraj	4	4.5	65	59	224.2	194.3	28.99	30.36
13	Subraman	6	5.4	29	34	210	207.5	13.80	16.38
14	Reymond	5	3.5	45.7	49	108.8	113.9	42	43.02
15	Merlin	8.6	7.5	84	76	297	267	28.28	28.46
16	Baby Girija	11	10.8	74.7	84.5	257.2	275	29.04	30.72
17	Thapamani	10	6	38	43.1	187	199.4	20.3	21.6
18	Ganeshan	30	27	56	40.5	171	170.2	32.7	23.79
19	Albin Jose	7	5	39	40.1	142.7	137.4	27.33	29.18
20	Marianesan	16	12	48	50.6	167.3	160.4	28.69	31.54

Annexure 6

Sl. No	Name	Serum ferritin (T0) (ng/mL)	Serum ferritin (T2) (ng/mL)	Hb(T0) (gm/dL)	Hb(T2) (gm/dL)	ESR(T0) (mm/Hr)	ESR(T2) (mm/Hr)	Serum albumin (T0) (gm/dL)	Serum albumin (T2) (gm/dL)
1	Ponnappan	188.4	106.3	8. 6	8.8	88	85	3.7	4
2	Masilamani	181.1	784.1	9.2	10.2	41	63	4.1	4.5
3	Devadhas	171	169	8.5	8.5	50	62	4	4
4	Vijayakumar	238	245.2	5.4	7	57	48	3.7	3.9
5	Nazeema	401.7	689.4	9.5	10.1	16	15	4.4	4.1
6	Vijayakumar	360	349	7.8	6.8	67	54	3.8	3.6
7	Mani	241.2	228	11.9	10.7	26	20	4.4	3.9
8	Natarajan	537.5	484.9	11.7	11.9	20	28	4.2	4
9	Justin	1278.5	1180	6.8	8.2	67	49	4	4.2
10	Brightdas	430	640.6	9.8	6	38	36	4.6	4.3
11	Aruldas	169.5	189	9.8	9.5	54	56	4.9	4.7
12	Kanakaraj	276	266	9.9	8.7	72	67	3.8	4
13	Subraman	1292.9	1240	9.7	10.2	69	64	4.5	4.2
14	Reymond	647	638	7.7	7.5	67	35	4.4	4
15	Merlin	347	366	10.1	11	77	68	3.7	3.6
16	Baby Girija	366	381	6.6	8.1	65	72	3.8	3.9
17	Thapamani	212	202.1	8.3	9.3	43	35	4.6	4.1
18	Ganeshan	251.2	267.5	8.7	6.6	75	117	3.9	4
19	Albin Jose	289	278	8.5	9.5	66	45	4	4
20	Marianesan	678	692.1	6.4	6.6	63	57	3.7	3.9

Annexure 6

Group 2(Haemodialysis>1 year)

Sl.No	Name	Serum creatinine (T0) (mg/dl)	Serum creatinine (T2) (mg/dl)	GFR(T0) (mL/ min/1.73m^2)	GFR(T2) (mL/min/ 1.73m^2)
1	Ponnappan	6.7	6.8	9	9
2	Masilamani	7.1	13.6	8	4
3	Devadhas	9.1	10	7	6
4	Vijayakumar	7.8	8.5	11	9
5	Nazeema	4.2	5.7	12	8
6	Vijayakumar	5.6	5.2	11	12
7	Mani	6.4	10.4	9	5
8	Natarajan	7.5	7	7	8
9	Justin	3	4.1	26	18
10	Brightdas	5.2	10.1	12	5
11	Aruldas	4	6.4	17	10
12	Kanakaraj	7.6	7.4	8	8
13	Subraman	3.1	3	21	21
14	Reymond	3.4	3.1	21	24
15	Merlin	10.1	9	5	5
16	Baby Girija	12	10.3	3	4
17	Thapamani	10.4	11.2	5	5
18	Ganeshan	3.2	3.5	19	17
19	Albin Jose	8.6	19.5	7	5
20	Marianesan	8.8	8.6	6	6

Annexure 6

Group 3(Chronic Periodontitis)

Sl NO	Name	CRP(T0) (mg/L)	CRP(T2) (mg/L)	Serum iron(T0) (µg/dl)	Serum iron(T2) (µg/dl)	Serum TIBC(T0) (µg/dl)	Serum TIBC(T2) (µg/dl)	TSAT(T0) %	TSAT(T2) %
1	Sandhya	2.4	1.8	63	93.1	194	231.3	32.47	40.25
2	Kaladevi	3.2	3.1	58	62	178.4	189	32.51	32.80
3	Ganesh	2.1	1.8	65.1	69	234	241.2	27.82	28.60
4	Nalini	2.2	1.4	49	54.5	234	264.3	20.94	20.62
5	Syamala	2.6	1.8	69.4	85.1	345	240	20.11	35.45
6	Ratheesh	2.7	2.2	77	72	197.2	186.3	39.04	38.64
7	Marybai	2.8	1.9	55.8	65	266.7	293.5	20.92	22.14
8	Annamma	3.5	3.1	50.3	53.4	278	265.4	18.09	20.12
9	Gopi	3.5	3.6	68.7	57.9	402.4	412	17.07	14.05
10	Shikha	2.6	2.1	75.4	78	306	321.4	24.64	24.19
11	Anandaroopan	1.9	1.5	64.3	64	248.5	261.3	25.87	24.49
12	Mary	1.2	2.1	80	82.7	215	227	37.2	36.43
13	Sreekumar	1.3	1	83.2	88.1	186	194	44.73	45.41
14	Pushpakaran	2.5	2	66.6	84.3	265.2	219.8	25.11	38.35
15	Suji	2.4	1.9	86	62.5	269	185	31.9	33.7
16	Vijila	2.3	1.6	89	92	280.2	274	31.76	33.5
17	Lekshmi	2.3	1.4	46.3	43	143.7	129.6	32.21	33.17
18	Saritha	3.2	2.4	79	74.5	196	180.3	40.30	41.32
19	Jayanthi	2.7	2.4	63	59	220	195	28.6	30.25
20	Jerald	2.6	2.8	72.6	70	165.2	157.8	43.94	44.35

Annexure 6

Sl. No	Name	Serum ferritin (T0) (ng/mL)	Serum ferritin (T2) (ng/mL)	Hb(T0) (gm/dL)	Hb(T2) (gm/dL)	ESR(T0) (mm/Hr)	ESR(T2) (mm/Hr)	Serum albumin (T0) (gm/dL)	Serum albumin (T2) (gm/dL)
1	Sandhya	21.2	15.6	14.1	13.2	4	4	4.2	4
2	Kaladevi	71	67.5	12	12.1	27	20	4.2	4.1
3	Ganesh	65.5	49	10	12.3	35	28	4	4.1
4	Nalini	21.3	19.2	10.9	12.7	20	14	3.9	4.7
5	Syamala	35.1	26	11	12.4	13	8	3.8	3.7
6	Ratheesh	65	55	10.2	11	19	13	3.9	3.6
7	Marybai	50.1	51.3	14.5	13.2	14	14	4.2	4
8	Annamma	79.3	72.9	10.4	10.4	29	22	4.2	4
9	Gopi	49.5	36	9.4	10.2	31	25	4.2	4.2
10	Shikha	34	31	12.4	11.4	17	22	4.1	4.3
11	Anandaroopan	22	16	11	12.1	19	25	4	4.2
12	Mary	61	54.7	13	12	9	14	4.4	4.1
13	Sreekumar	65	68	10.6	11.5	31	27	3.8	4
14	Pushpakaran	90	90.6	13.2	13	14	10	4.4	4
15	Suji	29.3	27	12.3	12.9	18	16	4	4
16	Vijila	38.1	40.3	12	13.3	17	17	4.3	4.1
17	Lekshmi	58	58	12.1	10	10	13	4	3.8
18	Saritha	83	89.6	11	13.4	24	17	3.8	4.2
19	Jayanthi	53.4	48.4	12.6	11.7	16	19	3.8	4.2
20	Jerald	36.5	31.2	11.5	12	27	21	4	4

Annexure 6

Group 3(Chronic Periodontitis)

Sl.No	Name	Serum creatinine (T0) (mg/dl)	Serum creatinine (T2) (mg/dl)	GFR(T0) (mL/ min/1.73m^2)	GFR(T2) (mL/min/ 1.73m^2)
1	Sandhya	0.8	0.8	101	101
2	Kaladevi	0.8	0.8	87	87
3	Ganesh	0.9	0.6	112	132
4	Nalini	0.7	0.7	108	108
5	Syamala	0.7	0.6	104	110
6	Ratheesh	0.6	0.6	122	122
7	Marybai	0.9	0.7	80	109
8	Annamma	1	0.9	64	72
9	Gopi	0.9	0.9	96	96
10	Shikha	0.8	0.7	96	113
11	Anandaroopan	0.9	0.7	117	129
12	Mary	0.9	0.8	80	93
13	Sreekumar	0.7	0.7	107	107
14	Pushpakaran	0.9	1	99	87
15	Suji	0.8	0.7	97	114
16	Vijila	0.6	0.7	116	110
17	Lekshmi	0.8	0.8	93	93
18	Saritha	0.9	0.8	87	100
19	Jayanthi	0.6	0.6	112	112
20	Jerald	0.7	0.7	113	113